43-647

ARCHIVES
OF
OPHTHALMOLOGY

EDITED IN ENGLISH AND GERMAN

BY

DR. H. KNAPP
OF NEW YORK

AND

DR. C. SCHWEIGGER
OF BERLIN

DR. W. A. HOLDEN
OF NEW YORK
ASSISTANT EDITOR

VOLUME XXII.

NEW YORK
G. P. PUTNAM'S SONS, 27 & 29 WEST 23D STREET
AND NEW ROCHELLE, N. Y.
LONDON: 24 BEDFORD STREET, STRAND
WIESBADEN: J. F. BERGMANN'S Verlag
PARIS: J. B. BAILLIÈRE, 19 Rue Hautefeuille
1893

COPYRIGHT, 1893
BY
BY G. P. PUTNAM'S SONS

Printed and Bound by
The Knickerbocker Press, New York
G. P. PUTNAM'S SONS

CONTENTS OF VOLUME XXII.

NUMBER I.

PAGE

1. Contributions to Clinical Ophthalmology: Being Gleanings from my Case-Books. By Swan M. Burnett, M.D., Ph.D., Washington, D.C. (With six illustrations) 1

2. Simple Method of Operating for Partial Tenotomy of the Recti Muscles (Graduated Tenotomy). By Eugene Smith, M.D. (With two wood-cuts) 16

3. The Diagnosis of Sympathetic Ophthalmia. By Dr. Leopold Weiss. Translated by Dr. Casey A. Wood, of Chicago 18

4. A Convenient Arrangement for the Temporary Use of Prisms. By William E. Baxter, M.D. 24

5. A Remarkable Case of Complete Paralysis of the Internus and Externus of Each Eye. By F. C. Hotz, M.D., Chicago . . 25

6. A New Perimeter. By J. Edward Giles, M.D. (With a wood-engraving) 28

7. A Case of Hemorrhagic Iritis, with Remarks. By Dr. Charles Zimmermann, Milwaukee, Wis. 31

8. History and Principles of Keratometry; Its Value and Limitation in the Correction of Astigmatism. By Carl Weiland, M.D. (With five drawings) 37

9. Congenital Ptosis with Synchronous Movements of the Affected Lid and Lower Jaw. By Alvin A. Hubbell, M.D. (With three illustrations) 65

10. A Case of Total Iris Absence. By L. D. Brose, M.D., Ph.D. . 70

11. A Case of Sympathetic Neuro-Retinitis with Plastic Iritis; Recovery; Microscopic Examination of the Enucleated Eye. By Dr. T. Y. Sutphen, Newark, N. J. 72

12. On Embolism of the Central Artery of the Retina. By Dr. Anton Elschnig. Translated and abridged by Dr. Ward A. Holden. (With three figures in the text, and two plates, Tab. II. and III. of vol. xxiv., German Edition) 74

PAGE

13. On Embolism of a Branch of the Central Artery of the Retina. By Dr. Ward A. Holden. (With five figures in the text) . . 90

14. On Traumatic Enophthalmus. By Dr. Theodor Beer, of Berne, Switzerland. Translated by Dr. Ward A. Holden. (With one figure in the text) 98

15. The Medico-Electric Eye-Bath in the Treatment of Scleritis and Episcleritis. By Dr. Joseph Norsa, of Rome. Translated by Dr. J. M. Mills, of New York 107

16. Further Communication on the Treatment of Trachoma. By H. Knapp. (With one wood-cut) 111

17. A Report of the Proceedings of the Twenty-Second Meeting of the Heidelberg Ophthalmological Society, held August 8, 9, and 10, 1892. By Prof. Karl Horstmann. Translated by Charles H. May, M.D., New York 119

18. Systematic Report on the Progress of Ophthalmology during the First Half of the Year 1892. By Dr. St. Bernheimer, in Vienna; Prof. Horstmann, in Berlin; and Dr. Silex, in Berlin. With the Co-operation of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. Deus, Berlin; Dr. Herrnheiser, Prague; Dr. Sulzer, Geneva; and others. Translated by Dr. Ward A. Holden and Dr. Casey A. Wood 125

A.—Dr. St. Bernheimer. General ophthalmological literature, 125; General pathology, diagnosis, and therapy, 127; Instruments and remedies, 128; Anatomy, 131; Physiology, 132.

B.—Prof. Horstmann. Errors of refraction and accommodation, 136; Lids, 138; Lachrymal apparatus, 142; Muscles and nerves, 143; Orbit and neighboring cavities, 145; Conjunctiva, cornea, sclera, anterior chamber, 146.

C.—Dr. P. Silex. Iris, 153; Choroid, 154; Glaucoma, 155; Sympathetic ophthalmia, 157; Vitreous, 158; Lens, 159; Retina and functional disturbances, 164; Optic nerve, 167; Injuries, foreign bodies, and parasites, 169; Ocular affections in constitutional disease, 172.

19. Book Reviews 177

The Relations of the Visual Organ and its Diseases to the Other Diseases of the Body and its Organs. (Die Beziehungen des Sehorgans, etc.) By Dr. Max Knies.
Les Maladies des Yeux dans leurs Rapports avec la Pathologie Générale. By Dr. Emile Berger.
Introduction into Ophthalmology. By Dr. J. Hirschberg.
A Manual of Clinical Ophthalmology. By H. F. Hansell, M.D., and J. H. Bell, M.D.
Ophthalmic Diseases and Therapeutics. By A. B. Norton, M.D.
Text-book of Ophthalmology. By Dr. E. Fuchs.
Transactions of the Ophthalmological Section of the Am. Med. Assoc.
A Handbook of the Diseases of the Eye and their Treatment. By H. R. Swanzy.

20. Miscellaneous Notes 181

Appointments.
Society Meetings.

NUMBER 2.

PAGE

1. Clinical Report of a Case of Temporal Hemianopsia with Recovery, Followed by Right Lateral Hemianopsia, Ophthalmoplegia, and Partial Recovery. By H. V. Würdemann, M.D., and J. S. Barnes, M.D., Milwaukee, Wis. (With seven perimetric double charts). 183
2. A Reply to Dr. C. E. Finlay's Criticism of my Investigations and Views on the Origin of Sympathetic Ophthalmia. By Robert L. Randolph, M.D. 194
3. A New Series of Test-Type. By Charles A. Oliver, M.D. . . . 200
4. Ophthalmia Nodosa, Caused by Penetrating Caterpillar Hair. By Dr. E. Krüger, of Bonn. Translated by Dr. Zimmermann, Milwaukee, Wis. (With Plate IV. of vol. xxiv., Germ. Ed.) . 202
5. On the Pathology and Pathogenesis of Retinitis Proliferans (Manz). By Dr. M. Banholzer. Translated by Dr. Ward A. Holden. (With Plate V. of vol. xxv., Germ. Ed.) 212
6. A Contribution to the Etiology of the So-called Retinitis Proliferans. By Dr. S. Schultze, Frankfort-on-the-Main. Translated by Dr. Ward A. Holden. (With two figures in the text) . . . 219
7. Treatment of Ptosis. By Dr. A. Meyer, of Wuerzburg. Translated by Dr. J. A. Spalding, Portland, Me. (With two illustrations in the text) 226
8. Observations of Contraction of the Visual Field According to Foerster's Type. By Dr. O. Koenig, of Breslau. Translated by Dr. J. A. Spalding, Portland, Me. (With twelve visual-field charts) 231
9. The Subject of the Designation of the Principal Meridians in Astigmatism before the Section on Ophthalmology in the College of Physicians of Philadelphia. By Dr. G. C. Harlan . . . 250
10. Systematic Report on the Progress of Ophthalmology during the Third Quarter of the Year 1892. By Dr. St. Bernheimer, in Vienna; Prof. Horstmann in Berlin; and Dr. Silex, in Berlin. With the co-operation of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. Deus, Berlin; Dr. Herrnheiser, Prague; Dr. Sulzer, Geneva, and others. Translated by Dr. Ward A. Holden . . . 262

    A.—Dr. St. Bernheimer. Text-books, monographs, and treatises on general, bibliographical, and historical subjects, statistical reports, 262; General pathology, diagnosis, and therapeutics, 264; Instruments and remedies, 264; Anatomy, 266; Physiology, 266.

    B.—Dr. Horstmann. Errors of refraction and accommodation, 268; Lids, 273; Lachrymal apparatus, 274; Muscles and nerves, 275; Orbit, 278; Conjunctiva, cornea, sclera, anterior chamber, 280.

11. Miscellaneous Notes 285

    Announcement of Books.
    Society Meetings.

NUMBER 3.

PAGE

1. On the Development and Extension of Orbital Sarcoma. By Dr. Ward A. Holden. (With six figures on a plate) 289

2. Bacteriological Investigations with Reference to the Origin of Sympathetic Ophthalmia. By Dr. Richard Greeff, of Frankfort-on-the-Main. Translated by Dr. Robert L. Randolph, of Baltimore, Md. (With three figures in the text) 298

3. Bilateral Hemianopsia, with Autopsy. By Prof. Schmidt-Rimpler, of Goettingen. Translated by Dr. J. A. Spalding, Portland, Me. (With two figures in the text) 313

4. The Position of the Source of Light and the Observer in Skiascopy or the Shadow Text. By Edward Jackson, A.M., M.D. . . . 323

5. Some Remarks upon Retinoscopy as a Method of Determining the Refraction of the Nucleus of the Lens. By John Dunn, M.D., Richmond, Va. 329

6. Contraction of the Visual Field in Traumatic Neurosis. By Dr. E. Fisher, of Dortmund. Translated by Dr. J. A. Spalding, Portland, Me. 334

7. Dislocation of the Lens into the Anterior Chamber, with Iridodialysis; Extraction; Recovery. By C. Zimmermann, M.D., Milwaukee, Wis. 337

8. Congenital Unilateral Anophthalmus. By C. Zimmermann, M.D., Milwaukee, Wis. 340

9. Extraction of Part of the Capsule as an Operative Procedure in Certain Cases of Secondary Cataract. By John Dunn, M.D., Richmond, Va. 344

10. A New Series of Test-Words for the Determination of the Power of Accommodation. By Charles A. Oliver, M.D. 347

11. Movements of the Upper Eyelid Associated with Lateral Movements of the Eyeball. By Dr. Harry Friedenwald, Baltimore, Md. 349

12. On Orbital Lower-Lid Cysts with Microphthalmus or Anophthalmus. By Dr. J. Mitvalsky. Translated by Dr. Ward A. Holden. (With plates VI. and VII. of vol. xxv. of German Edition) . 355

13. A New Phorometer. By A. E. Prince, M.D., Springfield, Ill. . 372

14. Systematic Report on the Progress of Ophthalmology during the Fourth Quarter of the Year 1892. By Dr. St. Bernheimer, in Vienna; Prof. Horstmann, in Berlin; and Dr. Silex, in Berlin. With the co-operation of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr.

PAGE

Deus, Berlin; Dr. Herrnheiser, Prague; Dr. Sulzer, Geneva, and others. Translated by Dr. Ward A. Holden . . . 375

A.—Dr. St. Bernheimer. General ophthalmological literature, 375; General pathology, diagnosis, and therapeutics, 377; Instruments and remedies, 378; Anatomy, 380; Physiology, 381.

B.—Prof. Horstmann. Errors of refraction and accommodation, 383; Lids, 385; Lachrymal apparatus, 387; Muscles and nerves, 387; Orbit and neighboring cavities, 389; Conjunctiva, cornea, sclera, anterior chamber, 391.

C.—Dr. P. Silex. Iris, 396; Choroid, 397; Glaucoma, 399; Sympathetic ophthalmia, 400; Vitreous, 402; Lens, 402; Retina and functional disturbances, 407; Optic nerve, 412; Injuries, foreign bodies (parasites), 415; Ocular affections in constitutional disease, 417.

15. Miscellaneous Notes 423

Book Review.
Appointment.
Prize Essay.

NUMBER 4.

1. On the Pathology of Hypopyon Keratitis. By Wilbur B. Marple, M.D. (With one figure in the text) 425

2. Some Remarks on the Refractive Value of any Two Cylinders or of Two Prisms Placed at Arbitrary Angles in the Trial Frame; with the Description of a New Instrument for Finding their Sphero-Cylindrical or Prismatic Equivalents, and also the Prismatic Effect of the Decentration of Lenses by a Graphic Construction without Calculation. By Carl Weiland, M.D. (With seven figures in the text) 435

3. Temperature-Topography of the Eye and Warm and Cold Applications. By Dr. P. Silex. Translated by Charles H. May, M.D., New York 450

4. Observations on Four Hundred and Six Cataract Extractions Performed by Prof. Haab, of Zurich. Collected by Dr. Theodor Arnold. Translated and abridged by Dr. J. A. Spalding, Portland, Me. 462

5. Glaucoma Experimentally Produced in Rabbits. By Prof. Richard Ulrich, of Strassburg. Translated by Charles H. May, M.D., New York 475

6. On Intraocular Cysts. By Dr. Richard Greeff, Frankfort-on-the-Main. Translated by Dr. Ward A. Holden. (With Plates VIII. and IX., vol. xxv., German edition) 481

7. New Colored-Letter Tests for Simulated Monocular Blindness. By James L. Minor, M.D., Memphis, Tenn. 493

8. Congenital Internal Occlusion of the Canaliculi; Treatment by Incision and Probing Successful. By Alanson J. Abbe, M.D., Fall River, Mass. 496

9. Single-Suture Operation for Advancing the Rectus. By A. E. Prince, M.D., Springfield, Ill. (With two figures in the text) . 498

PAGE

10. On the Visual Acuteness of the Periphery of the Retina, and on a New Method of Determining it. By Dr. Arthur Groenouw. Translated by Dr. Ward A. Holden. (With two figures in the text) 502

11. Systematic Report on the Progress of Ophthalmology during the First and Second Quarters of the Year 1893. By Dr. St. Bernheimer, in Vienna; Prof. C. Horstmann, in Berlin; and Dr. Silex, in Berlin. With the Co-operation of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. Deus, Berlin; Dr. Herrnheiser, Prague; Dr. Sulzer, Geneva; and others. Translated by Dr. Ward A. Holden . . . 519

A.—Dr. St. Bernheimer. General ophthalmological literature, 519; General pathology, diagnosis, and therapy, 522; Instruments and remedies, 525; Anatomy, 527; Physiology, 528.

B.—Dr. Horstmann. Anomalies of refraction and accommodation, 530; Lids, 534; Lachrymal apparatus, 535; Muscles and nerves, 536; Orbit and accessory cavities, 543; Conjunctiva, cornea, sclera, anterior chamber, 547.

12. Miscellaneous Notes 555

I.—A Text-Book of Ophthalmology. By Wm. F. Norris, A.M., M.D.
II.—A Handbook of Ophthalmic Science and Practice. By Henry E. Juler, F.R.C.S.
III.—A Dictionary of Medical Science. By Robley Dunglison, M.D., LL.D.
IV.—Études sur les Tumeurs de l'Œil, de l'Obite et des Annexes. By Dr. Félix Lagrange.
V.—Embryology of the Eye. By Ward A. Holden, A.M., M.D.

13. Index 559

ARCHIVES OF OPHTHALMOLOGY

NOTICE TO OUR READERS.

The ARCHIVES OF OPHTHALMOLOGY contain *original papers* on all branches of ophthalmic surgery, and *original reports* on the progress of ophthalmology. The original papers occupy from two thirds to three fourths of the space, and their scope embraces all subjects of scientific and practical interest connected with this department of medicine.

The object of the ARCHIVES is to guard, solidify, extend, and diffuse the stock of acquired ophthalmic knowledge by recording the constant addition of new observations, investigations, methods and means of examination, clinical experience, instruments and remedies, and further by taking part in the discussion of the questions of the day with a spirit alike conservative and progressive.

Particular attention is paid to the preparation of the Report on the Progress of Ophthalmology. The report is intended to furnish a *complete, systematic, and early* review of the current ophthalmological literature of the world. It has been necessary to divide the work of its preparation among a number of collaborators according to the following arrangement:

DR. ST. BERNHEIMER, of Vienna.

1. *General Ophthalmological Literature:*
   - *a.*—Text-books (not abstracts, but notes pointing out what is new and characteristic).
   - *b.*—Monographs and papers on historical, statistical, and general subjects; reports on societies and hospitals.
   - *c.*—Periodicals on our specialty (short notices of their character, time and place of publication, the titles of the original papers, with the names of their authors).

2. *General Pathology, Diagnosis, and Therapeutics*, including new instruments, apparatus, and remedies.
3. *Normal Anatomy and Physiology*, including color-blindness.

Dr. C. HORSTMANN, Prof. at the University of Berlin (Potsdamer Strasse 4):

1. *Anomalies of Refraction and Accommodation.*
2. *Eyelids.*
3. *Lachrymal Apparatus.*
4. *Muscles and Nerves.*
5. *Orbit and Adjacent Cavities.*
6. *Conjunctiva, Cornea, Sclerotic.*
7. *Injuries and Foreign Bodies of these parts.*

Dr. P. SILEX, Lecturer at the University of Berlin.

1. *Uveal Tract*, including glaucoma.
2. *Refracting Media*, including the lens.
3. *Retina and Optic Nerve.*
4. *Injuries and Foreign Bodies of these parts.*
5. *Ocular affections in connection with general diseases.*

In order to obtain complete and recent reviews on the non-German ophthalmological literature, special reporters have been selected for different countries, who will send monthly abstracts of all local ophthalmological publications to Dr. C. Horstmann, of Berlin, the editor of the Report. Dr. Horstmann will distribute these abstracts among the German reporters according to the above plan, in order to preserve the systematic arrangement which we think of paramount importance.

The special reporters are:

For America: Dr. SWAN M. BURNETT, 1770 Massachusetts Ave., Washington, D.C.

For Great Britain and Ireland: Dr. L. WERNER, 27 Upper Merrion Street, Dublin.

For France and Belgium: Drs. D. E. SULZER, Geneva, and P. VON MITTELSTÄDT, Metz.

For Germany: Drs. BERNHEIMER, Vienna, HORSTMANN, SILEX, and DEUS, in Berlin.

For Italy: Dr. DANTONE, Rome.

For Russia: Prof. HIRSCHMANN, Charkow.

For Scandinavia: Drs. OLE BULL and SCHIÖTZ, Christiania.

For Holland: Dr. C. A. H. WESTHOFF, of Amsterdam.

In the interest of our readers, who are scattered over the whole world—and whom, like ourselves, we desire to keep informed of all advances in ophthalmology,—furthermore in their own interest, authors of ophthalmological works are requested to send reprints, duplicates of journals, or copies for reviewing, to one of the above-named reporters, or to the editors or publishers of the Archives, according to their convenience.

The abstracts of American ophthalmological literature formerly appeared under a special head, but are now embodied in the systematic Report. Text-books may be specially noticed.

The number of good papers offered us for publication has increased to such an extent that it is no longer possible to translate all of them unabridged. Whenever a German paper is condensed in the English version, or *vice versa*, it will be so stated. Should any one of our readers wish to recur to the original, the editors will always take pleasure in sending it for reference.

Under the heading of "Miscellaneous Notes," there will be published all kinds of professional news which specially concern the oculist, *e. g.*, appointments, honors, resignations, vacancies, new ophthalmic hospitals, opportunities for instruction, prize questions and essays, announcement of Society meetings, etc. Brief notices of this kind will be thankfully received by the editors.

Original papers of value from any source will, as heretofore, be welcome to the ARCHIVES, and are solicited.

EDITORS:

H. KNAPP, M.D.,
26 WEST 40TH STREET, NEW YORK.

C. SCHWEIGGER, M.D.
ROONSTR. 6, BERLIN.

NOTICE TO CONTRIBUTORS.

The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with their contributions.

1. As original communications the ARCHIVES can accept only such papers as have never been printed nor are intended to be printed in other journals. If a preliminary communication on the subject of a paper has been published, the author is requested to state this in the letter accompanying his manuscript. It is understood that contributors to these ARCHIVES and editors of other periodicals will make no abstracts of the original papers published in this journal without giving it due credit for the same.

2. Authors will receive gratuitously twenty-five reprints of their articles. If a greater number is desired,—notice of which should be given at the head of the manuscript,—only the additional cost of presswork and paper will be charged to the author.

3. In preparing manuscript for the compositor it is requested that the following rules be adhered to:

a. Write on one side of the paper only.

b. Write without breaks, *i. e.* do not begin a new sentence on a new line. When you want to begin a new line or paragraph at a given word, place before it in your MS. the sign ¶.

c. Draw a line along the margin of such paragraphs as should be printed in smaller type—for instance, all that is clinical history in reports of cases, etc.

d. Words to be printed in *italics*, should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times.

4. Authors may receive proofs for revision if they will kindly return them without delay. We beg however to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them, to make, if possible, no alterations at all in their MSS., or, at least, to limit these to what is of essential importance.

CONTENTS OF VOLUME XXII., NUMBER 1.

		PAGE
1.	Contributions to Clinical Ophthalmology: Being Gleanings from my Case-Books. By Swan M. Burnett, M.D., Ph.D., Washington, D.C. (With six illustrations)	1
2.	Simple Method of Operating for Partial Tenotomy of the Recti Muscles (Graduated Tenotomy). By Eugene Smith, M.D. (With two wood-cuts)	16
3.	The Diagnosis of Sympathetic Ophthalmia. By Dr. Leopold Weiss. Translated by Dr. Casey A. Wood, of Chicago	18
4.	A Convenient Arrangement for the Temporary Use of Prisms. By William E. Baxter, M.D.	24
5.	A Remarkable Case of Complete Paralysis of the Internus and Externus of Each Eye. By F. C. Hotz, M.D., Chicago	25
6.	A New Perimeter. By J. Edward Giles, M.D. (With a wood-engraving)	28
7.	A Case of Hemorrhagic Iritis, with Remarks. By Dr. Charles Zimmermann, Milwaukee, Wis.	31
8.	History and Principles of Keratometry; Its Value and Limitation in the Correction of Astigmatism. By Carl Weiland, M.D. (With five drawings)	37
9.	Congenital Ptosis with Synchronous Movements of the Affected Lid and Lower Jaw. By Alvin A. Hubbell, M.D. (With three illustrations)	65
10.	A Case of Total Iris Absence. By L. D. Brose, M.D., Ph.D.	70
11.	A Case of Sympathetic Neuro-Retinitis with Plastic Iritis; Recovery; Microscopic Examination of the Enucleated Eye. By Dr. T. Y. Sutphen, Newark, N. J.	72
12.	On Embolism of the Central Artery of the Retina. By Dr. Anton Elschnig. Translated and abridged by Dr. Ward A. Holden. (With three figures in the text, and two plates, Tab. II. and III. of vol. xxiv., German Edition)	74

PAGE

13. On Embolism of a Branch of the Central Artery of the Retina. By Dr. Ward A. Holden. (With five figures in the text) . . 90

14. On Traumatic Enophthalmus. By Dr. Theodor Beer, of Berne, Switzerland. Translated by Dr. Ward A. Holden. (With one figure in the text) 98

15. The Medico-Electric Eye-Bath in the Treatment of Scleritis and Episcleritis. By Dr. Joseph Norsa, of Rome. Translated by Dr. J. M. Mills, of New York 107

16. Further Communication on the Treatment of Trachoma. By H. Knapp. (With one wood-cut) 111

17. A Report of the Proceedings of the Twenty-Second Meeting of the Heidelberg Ophthalmological Society, held August 8, 9, and 10, 1892. By Prof. Karl Horstmann. Translated by Charles H. May, M.D., New York 119

18. Systematic Report on the Progress of Ophthalmology during the First Half of the Year 1892. By Dr. St. Bernheimer, in Vienna; Prof. Horstmann, in Berlin; and Dr. Silex, in Berlin. With the Co-operation of Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. Deus, Berlin; Dr. Herrnheiser, Prague; Dr. Sulzer, Geneva; and others. Translated by Dr. Ward A. Holden and Dr. Casey A. Wood 125

A.—Dr. St. Bernheimer. General ophthalmological literature, 125; General pathology, diagnosis, and therapy, 127; Instruments and remedies, 128; Anatomy, 131; Physiology, 132.

B.—Prof. Horstmann. Errors of refraction and accommodation, 136; Lids, 138; Lachrymal apparatus, 142; Muscles and nerves, 143; Orbit and neighboring cavities, 145; Conjunctiva, cornea, sclera, anterior chamber, 146.

C.—Dr. P. Silex. Iris, 153; Choroid, 154; Glaucoma, 155; Sympathetic ophthalmia, 157; Vitreous, 158; Lens, 159; Retina and functional disturbances, 164; Optic nerve, 167; Injuries, foreign bodies, and parasites, 169; Ocular affections in constitutional disease, 172.

19. Book Reviews 177

The Relations of the Visual Organ and its Diseases to the Other Diseases of the Body and its Organs. (Die Beziehungen des Sehorgans, etc.) By Dr. Max Knies.

Les Maladies des Yeux dans leurs Rapports avec la Pathologie Générale. By Dr. Emile Berger.

Introduction into Ophthalmology. By Dr. J. Hirschberg.

A Manual of Clinical Ophthalmology. By H. F. Hansell, M.D., and J. H. Bell, M.D.

Ophthalmic Diseases and Therapeutics. By A. B. Norton, M.D.

Text-book of Ophthalmology. By Dr. E. Fuchs.

Transactions of the Ophthalmological Section of the Am. Med. Assoc.

A Handbook of the Diseases of the Eye and their Treatment. By H. R. Swanzy.

20. Miscellaneous Notes 181

Appointments.

Society Meetings.

VOL. XXII. No. 1.

ARCHIVES OF OPHTHALMOLOGY.

CONTRIBUTIONS TO CLINICAL OPHTHALMOLOGY: BEING GLEANINGS FROM MY CASE-BOOKS.

By SWAN M. BURNETT, M.D., Ph.D., Washington, D. C.

(*With six illustrations.*)

(*Continued from p. 262, vol. xxi.*)

EYE AFFECTIONS FOLLOWING LA GRIPPE.

To the literature of eye affections following epidemic influenza, I can contribute the following cases:

Case X.

RETROBULBAR NEURITIS IN A NEGRO FOLLOWING LA GRIPPE, SUCCEEDED BY ATROPHY AND GREAT NARROWING OF THE VISUAL FIELDS.

Taylor Triplet, a colored man, twenty years of age, was seen at my clinic first on June 6, 1891. He had had an attack of *la grippe* two months before, and on convalescence had noticed that something was wrong with his eyes. He had at the time a central vision = $\frac{1}{5}$ in either eye. Visual fields as in diagrams Fig. 9 A and B. Inside of this field color perception was good.

The ophthalmoscope revealed nothing abnormal, except, perhaps, a slight blanching of the nerves at the upper outer quadrants. The patella reflex is not good, but he stands well with his eyes shut, and can walk backwards. There is no pain in the head, and he does not remember that there was any unusual pain in the eyes or orbits during the attack of influenza. His intellectual faculties are the same as before the attack. No history of syphilis can be elicited.

He was not seen again until November 16th, when he was led to the clinic, not being able to find his way about alone. His

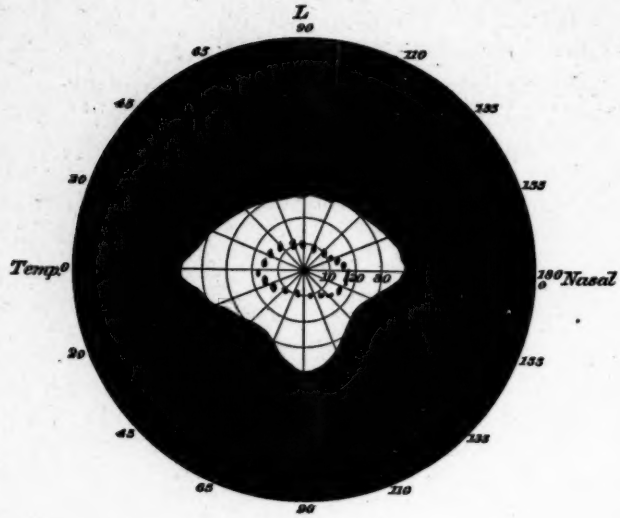

L
90
65
110
45
135
20
155
Temp.
0
180
Nasal
20
155
45
135
65
110
90

FIG. 9.—A.

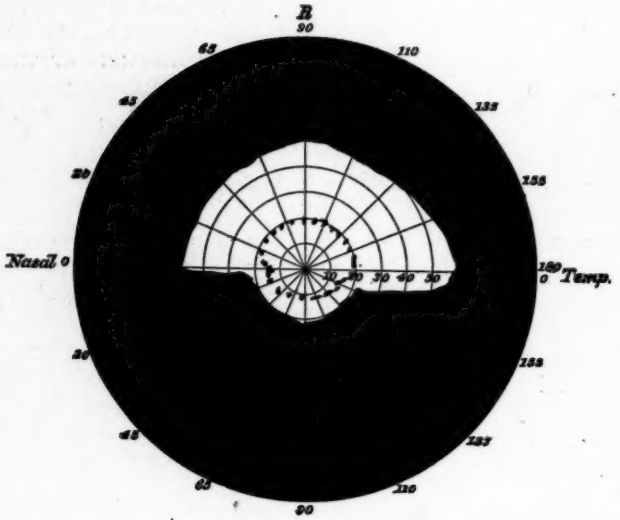

B
90
65
110
45
135
20
155
Nasal 0
180
Temp.
20
155
45
135
65
110
90

FIG. 9.—B.

visual fields were then contracted to the inner dotted circle in the diagrams. Central vision was $\frac{6}{12}$ L, $\frac{6}{20}$ R, and colors were correctly distinguished. Both disks were now white, but the retinal vessels were normal as to number and size. There were some capillaries on the disk surfaces. On December 3d the fields had contracted to less than 10° in both eyes, but he could still distinguish single letters of No. 18 at 5 *m.* Color perception within this limit was good. He disappeared and has not been seen since.

Case XI.

RETROBULBAR NEURITIS, ATROPHY. LATERAL DIPLOPIA, CURED.

James Eastman, a negro of thirty, was taken with *la grippe* on November 30, 1891. The attack was rather severe. About Christmas-time he noticed trouble with his eyes, preceded by some pain in the back of the head. There was lateral diplopia and dimness of vision. The diplopia ceased to be noticeable in March, but the failure in vision continued to increase until, at the time of examination (June 10, 1892), there was barely perception of light. The pupils are of medium size and respond feebly to light. Movements of the eye and lids good; media clear. The disks are white and rather dirty-looking; outlines not very sharp. Retinal arteries much narrowed in calibre; veins about normal in size and not tortuous. There is quite a noticeable white streak along each side of all the large vessels, which extends even on to the surface of the disk. In the right eye some small white dots and oblong patches are seen to the lower and outer side of the disk, mostly along or in the vicinity of the blood-vessels. The patient's general health is, and has always been, good, and there are no signs of ataxia.

Case XII.

ATROPHY OF BOTH OPTIC NERVES FOLLOWING LA GRIPPE.

Smith Jackson, colored, age twenty-eight, had influenza in February, 1891. A month afterwards he noticed that vision was failing, and the failure has been gradually increasing until now (four months after the first symptoms were noticed) he counts fingers at three feet with either eye. The visual fields are much contracted concentrically. The disks are white, with good sharp outlines, and the retinal vessels are but little, if any, changed. The tendon reflex is good.

Case XIII.

CENTRAL SCOTOMA IN ONE EYE FOLLOWING LA GRIPPE.

Mrs. S. E. J——, aged forty, seen first May 26, 1891. Had *la grippe* in the early spring, and on convalescence noticed that vision in the right eye was bad, and that there was a "spot" before it, through which objects look blurred and oftentimes of a reddish color. Vision in that eye = $\frac{5}{24}$, and, under a mydriatic, was increased to $\frac{5}{18}$ by a + 0.75. Fundus seemed normal. She also has homonymous lateral diplopia at the extreme left upper field of fixation. The scotoma has a diameter of 5 *cm* at a distance of 50 *cm*. The visual field is slightly contracted. She was under observation for a month, during which time no change in the condition was noticed.

Besides acting directly and primarily as a cause of neuritis, it would seem that the poison of influenza also hastens the operation of other causes of neuritis, as it does of those of other diseases, tuberculosis in particular. The two following cases would seem to be instances in point.

Case XIV.

COMPLETE ATROPHY OF BOTH OPTIC NERVES IN A MAN WHO HAD BEEN A HEAVY SMOKER, AFTER AN ATTACK OF LA GRIPPE.

J. H. M——, thirty-eight years of age, seen for the first time November 14, 1891. Nine months before he had had an attack of influenza in which the pain was mostly over the left eye, and immediately after noticed a decided failure of vision in the left eye, which has gradually increased until he can now count fingers only at 2 *m*. Vision in L eye, $\frac{5}{12}$.

Six years ago he had a paralysis of the left external rectus, after a blow on the left side of the head, which disappeared in the course of seven months. He had some diplopia again on recovering from *la grippe*. There is no history of specific infection. His wife has had four children; one died of small-pox, another of meningitis, the two living are healthy. The V F in left is contracted; in the right it is about normal. In the left the optic disk is grayish-white, and slopingly excavated all around. There are but few vessels on the disk. In the R the atrophy

and excavation are confined to the outer side, the inner side being on a level with the fundus. With the left he recognizes blue and green, but confounds red and light yellow. Colors in right normal. He has been a great consumer of cigars, smoking often as many as thirty daily. At time of attack smoked regularly six pipes a day. He was seen again July 16, 1892, and in the meantime had seen surgeons in New York and Philadelphia, who had treated him with strychnia and bichloride. He had gotten steadily worse, so that now there is only perception of light in left, and in right V = $\frac{3}{60}$. Left pupil widely dilated and immovable. The right disk is bluish-white and slopingly excavated; vessels do not appear to be greatly changed.

Case XV.

FAILURE OF VISION AFTER LA GRIPPE IN A MAN WHO SMOKED RATHER EXCESSIVELY.

Mr. A. P. F——, age fifty-six. Had an attack of influenza two years ago, complicated with pneumonia. In November, 1891, he had another, in which the gastric and intestinal symptoms were most prominent, and, being in the South at the time, he took 100 grains of quinine in ten days. On January 1, 1892, he observed that his vision was failing. There was no pain in the eyes, but he noticed flashes of light when he was in the dark. At the time of examination, March 6, 1892, vision = $\frac{5}{60}$ in either eye, with + 2 = $\frac{5}{24}$; but all objects have blurred outlines. Visual fields for white somewhat contracted concentrically, as were also those for colors. Central color perception good; no specific history could be elicited, and the tendon reflex was good. There was no change in the fundus which could satisfactorily account for the diminished vision. The disks were of good color and the retinal vessels were normal. He was put upon strychnia, $\frac{1}{60}$ gr. three times a day, and this was continued for a month. At the end of that time there was no essential improvement. It was then learned for the first time that he smoked more than moderately, sometimes as many as eight or ten cigars a day. His smoking was stopped, and in the course of several days an improvement was noted. This continued, with occasional slight relapses, until it reached almost the normal when last seen some four months after the first visit.

Case XVI.

SUDDEN BLINDNESS IN LEFT EYE AFTER LA GRIPPE.

Chas. F——, colored, age thirty-one, seen first May 12, 1891. Had *la grippe* this spring, and on convalescence noticed one day that he was blind in the left eye. During his illness he had great pain in the left temple, which radiated towards the left ear. In this eye the visual field is entirely lacking, except at the upper and inner part. The media are clear; the disk is paler than its fellow and slightly excavated. The other eye is normal.

I have notes of a number of cases of paresis of the various ocular muscles, which showed itself as a succedaneum of influenza. Sometimes but one muscle was attacked, but frequently two or more. In the majority of cases these had a happy issue in complete recovery.

There have been, in addition to these, a number of cases of heterophoria with asthenopia and other nervous symptoms, in which the trouble as regards the eyes and head dated from an attack of the disease.

Case XVII.

HEREDITARY CONGENITAL OPTIC-NERVE ATROPHY.

Cases of this peculiar affection are not so commonly met with as to make the report of other instances without interest, particularly in view of the fact that we are not yet certain that all the cases should be placed in the same category. Many of those reported have been instances of optic neuritis, some of them coming on a number of years after birth. In the cases here reported it would seem that, some at least, of those afflicted were born with the amblyopia upon them, and we are not able in such a case to say how far arrest in development may have operated as a cause. The presumption, however, seems to be in favor of a neuritis, possibly intrauterine. The fact that it is hereditary and that the neuritis appears for the most part in young people would seem to show that the developmental processes are at least defective and play an important *rôle* in all cases.

Scott Holt, aged twelve, was seen for the first time on May 1, 1892. He was sent to me from the country by his physician in order to have glasses fitted for the improvement of his vision, which was very bad. On examination I found that he could count fingers only at one metre's distance with either eye, and, according to the statement of his mother who accompanied him, he had never seen any better. There was a white atrophy of both disks, but no marked excavation. The retinal vessels were normal in number and size. The visual fields were much contracted for form. In testing with Holmgren's worsteds he mixed grays and blues with green, and the browns and olives with pink. The teeth were good and there was no evidence of inherited disease. The ophthalmoscope showed emmetropia.

Case XVIII.

OPTIC-NERVE ATROPHY IN THE MOTHER OF THE FORMER PATIENT AND HER FAMILY.

After the examination of the son was finished the mother informed me that her vision was also defective, and on testing I found it to be $\frac{5}{60}$ in either eye. The optic disks were bluish-gray, but of good outline. The retinal vessels were normal. Visual fields contracted for form. Color-test gave a mixing of gray and blue with green ; salmon, yellow, green, and purple with pink ; purple and green with blue. She does not remember that her vision has ever been better than it is now.

On inquiry I learned that very defective vision was common among her relations on her mother's side. Her mother's vision had been about the same as her own, and her maternal grandfather also saw very badly ; and in all there were five uncles and aunts on her mother's side who were known to have had impaired sight, and, presumably, due to the same cause. In her father's family, as far as known, there were no instances of markedly bad vision.

TENONITIS AND ORBITAL CELLULITIS.

While orbital cellulitis in not an uncommon affection, it is not of so frequent occurrence as to render some points of the following cases devoid of interest.

Case XIX.

TENONITIS.

I was asked by Dr. Mauss to see with him a girl of eleven years on the 2d of March, 1891. He had been called to her the day previous and learned that three days before she had complained of a slight headache and the parents noticed a swelling of the right eyelids. She had been in good health and no cause could be assigned for the trouble. There was no history of a blow or any traumatic injury. There was, when I saw her, a marked exophthalmos directly forward, some slight swelling and redness of the lids, and a chemosis of the ocular conjunctiva, quite pronounced below. The globe was absolutely fixed, there being not the slightest movement in any direction; pupil dilated and immobile; vision much reduced, and some slight pain at times on that side of the head. Temperature normal. The media were clear, but the sharp outlines of the disk were lost. The retina looked swollen, grayish, and infiltrated; no redness distinguishable in the fundus; veins a little swollen but not very tortuous; no hemorrhages. On the 3d I made punctures, under chloroform, into the orbit, through the lids above the inner canthus and between the outer canthus and the chemotic conjunctiva, going by the side of and back of the globe. Bloody serum escaped, but no pus. Temperature still normal. On the 4th the ophthalmoscopic picture was the same, but there was a slight movement of the globe up and down. On the 5th there was greater movement, and a flow of tears, for the first time since the attack. The fundus appeared somewhat red and more vessels were visible. Mercurial inunctions were ordered and a blister applied to the temple. On the 7th the exophthalmos had perceptibly diminished, but there was still no movement outward. On the 9th, the retinal vessels were much larger and the outlines of the disk were faintly seen and there was a slight movement of the ball outward. On the 11th the disk was clearer, but the movement outward was still very slight. Improvement continued steadily, so that on the 19th, when she was examined at the office, vision in the right (affected) eye was $\frac{5}{18}$, that in the left being $\frac{5}{5}$; pupil smaller and responsive to light; disk quite clear in outline and only a slight tortuosity of the vessels. Ophthalmometric examination of the cornea showed nothing different from that of the fellow eye. 29th, diplopia in the outer fields of fixation, vision right $\frac{5}{8}$. April 12th,

diplopia only at the extremes of fixation fields; vision ⅘. Sept. 18th, an exophoria of 2 P. D. both for near and far.

It is claimed that we can draw a clear line of distinction between orbital cellulitis and tenonitis, and if we can, this case would certainly fall, I think, in the category of tenonitis, and would be of the serous form. In tenonitis we would have the pressure closer around the globe, involving all the muscles, producing absolute immobility and the exophthalmos would be directly forward. The pressure on the nerve and its vessels would also be more direct than in the case of abscess, unless the abscess were situated in its immediate vicinity. The cause of the inflammation in this instance I cannot even surmise. Such cases have been referred to *la grippe*, but this case occurred before the epidemic of *la grippe* appeared among us in 1891. It made its appearance that year in April, and, besides, there was at no time any febrile or other systemic disturbance; the trouble remained purely local. It is not without interest to note the complete recovery of the retina from a very intense serous infiltration, and also the entire absence of hemorrhages, which are so commonly an accompaniment of mechanical obstruction to retinal circulation.

Quite distinct from this case in its clinical aspects is the following, which is one of ordinary cellulitis, probably metastatic, ending in abscess.

Case XX.

ORBITAL ABSCESS.

A man of twenty-five years was admitted to Garfield Hospital for a fever supposed to be malarial. He also had a stricture of urethra which he was waiting an established convalescence to have treated. On the 19th Dec., 1891, he complained of pain in the upper maxilla of the right side, and within ten hours there was a great swelling of the lids and an exophthalmos somewhat downward and outward. The eye was very tender to the touch, conjunctiva was chemotic below, and there was a slight discharge of muco-pus. Movement of the eye limited in all directions. Temperature reached 101° that day. The pupil had been dilated by

atropine, the fundus was normal, and the vision good. On the 22d, a painful swelling appeared on the left wrist. There was no indication of a pointing of an abscess in the orbit; temperature from 99° to 101°; treatment, hot applications externally. I was unable to see him again until the 26th, when I found that the abscess had pointed above the inner canthus and discharged a quantity of pus. It was kept empty by pressure on the globe backward and inward. On the 29th I opened another abscess pointing below the inner canthus. The movements of the eye good and vision unimpaired. On Jan. 20, 1892, the sinus had healed and the eye was in a normal condition. The abscess at the wrist was still discharging.

Case XXI.

TWO ATTACKS OF DOUBLE PAPILLITIS IN THE SAME PATIENT.

I much regret that the notes of the following case, recorded at the time, were lost, but the essential facts were as follows:

A colored woman of thirty was seen at my clinic for the first time about seven years ago. She had then very marked double papillitis and the vision was very much impaired though she could see to get about alone. The only complaint she made at that time was of headache, which was quite steady and had been for some time. There was no paresis or paralysis. Her intelligence was of a low order, and a clear clinical history could not be obtained. There were no signs of central nervous disease discoverable aside from that above stated. Thinking that specific disease might be at the bottom of the trouble, though the history pointed very indefinitely in that direction, she was put on specific treatment, and in the course of some weeks the papillitis began to subside. After four or five months there was scarcely a trace of it left, and her vision was vastly improved. Three years later she returned, and there was a full-blown papillitis in both eyes, accompanied by a more serious impairment of vision than before. She was unable to go about alone. There was no paralysis, but her mental condition had deteriorated sadly, and she complained of an incessant headache. She was not able to give any intelligent account of herself or her symptoms, and in a short time departed and we were unable to find her.

I have on more than one occasion seen papillitis, which was due undoubtedly to intracranial trouble, subside, but, so far as my own experience goes or my examination of the literature allows me to judge, cases of a second attack must be very rare, though I see no reason *a priori* why they should be. The intracranial conditions which lead to the papillitis are, as we know, very fluctuating, and the cause which leads to one attack would certainly be liable, when renewed, to bring about the conditions for the renewal of the neuritis.

Case XXII.

GUMMATA OF THE CILIARY REGION.

Two reasons have led to the reporting of the following cases. The first is that instances where gummata of the eye have come to enucleation and examination, though there are several cases on record, are not so common as to render further contributions to their study altogether superfluous, since the manifestations of syphilis in the eye is always a matter of serious moment when the possibility of its issue is considered. The second is the fact of the rapidity and malignancy with which these growths developed, and particularly when this is taken in connection with their appearance in negroes, in whom all syphilitic manifestations are much milder and more easily controlled than in the white race. The latter statement as to the mildness of syphilis in the negro may be new to some, but it is one which is supported by my own experience in an eye-clinic in which three fourths are of the African race, and by the experience of my surgical friends whose practice is among the same class. The only explanation of this that I have heard offered is that this race, through its proneness to suppuration at the initial lesion or at the glandular implications, either absorbs less virus or throws it off more readily than does the white race. On this point, however, I think we are in need of more substantiating proof, for my own observations will not allow me to grant a greater proneness to suppuration, at least in the eyes of the negro, either from inflammation or after operation.

Joseph S——, colored, aged twenty-three, came to the clinic for the first time on November 2, 1888. He then had a simple iritis of the right eye. He gave a history of an initial lesion some four months previously. There was no eruption, and he said there had been no considerable enlargement of the inguinal glands. The post-cervical glands were swollen. He was put on the usual specific mixture, and ordered atropine drops. On the 15th a gumma was noticed on the iris at the outer lower pupillary edge. The dosage of the specific mixture was increased, but the increase in the inflammatory process was so rapid that he was sent to Garfield Hospital for better care and more vigorous treatment. On the 28th an hypopyon appeared and the general condition of the eye was so much worse that he was put upon a vigorous course of deep muscular injections of sublimate and inunctions. In spite of this, on the 30th, a swelling 3 *mm* long made its appear-

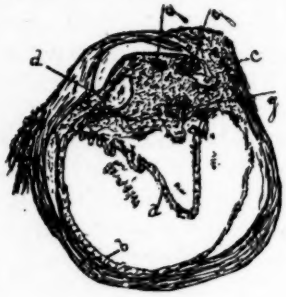


FIG. 10.

ance in the upper ciliary region. The injections and inunctions were continued, but with no apparent effect on the eye, which was frightfully painful and required the constant use of opiates for relief. The swelling increased slowly, and the eye took on a generally disorganized look. The patient appealed so persistently for a removal of the eye on account of the pain that I acceded to his request and the eye was enucleated on the 22d of December.

The eye after hardening in Müller's fluid was cut by Dr. Wm. Gray, of the Army Medical Museum.

An antero-posterior section through the ball of the eye is given in Fig. 10. The sclera is intact except at the ciliary region where the growth has pushed its way through both it and the cornea at *c*. The cornea and sclera on the other side seem in a

fairly normal condition. The growth has thrust itself inward to the back of the lens, *d*. The section is through the middle of the growth, and consequently the lens is cut near its equator, which has been somewhat dislocated to the other side. The ciliary body on this side also participates to an extent in the morbid process. The choroid, *b*, is detached except at the posterior part, and the retina, *a*, is wholly separated and drawn far forward, and is implicated, in its anterior portion, with the growth. The growth itself is not uniform in structure, but presents areas, *g*, of greater density than the surrounding parts and which are quite clearly circumscribed.

FIG. 11.

A magnified appearance of a portion where it passes out through the cornea is shown in Fig. 11.

The sclera, *s*, is seen to be in fairly normal state, and so is the cornea, *c*, immediately adjoining. At least they are not involved in the special pathological process. The mass is composed entirely of small round cells with only a few connective-tissue fibres. At the spots of greater condensation, *g*, they are so far reduced in size as to be almost amorphous, and are evidently the beginnings of a process of breaking down. In these there is no connective tissue whatever. Macroscopically these

spots have an appearance not unlike the giant cells of tubercle. The larger part of the mass itself is not vascular, but in the degenerated corneal and in the choroidal and ciliary tissue there are numbers of vessel, *v*, in all of which there is abundant evidence of a thickening of the vascular walls. In fact, all the vessels wherever seen show signs of a vasculitis. The capsule of the lens, *l*, is intact.

The other eye was not attacked, and when the patient was seen for the last time some months after the enucleation, there was no evidence of the disease apparent.

Case XXIII.

H. J., colored, aged eighteen, was admitted to the clinic January 4, 1889, with the diagnosis of specific iritis of the left eye. He said that he contracted the initial lesion in July, 1888, and was treated at the Freedman's Hospital for secondary eruption in September. In November the eye was inflamed for the first time. Since then it has been painful at times and latterly almost continuously. There were the signs of an iritis of rather unusual severity, and he was put upon strong specific treatment. At the end of a week a swelling was noticed at the upper ciliary region, and he was sent at once to the Garfield Hospital and submitted to the same treatment by inunctions and deep muscular injections of bichloride as in the preceding case, but with as little avail. The pain complained of by these patients was most excruciating and continuous, and this one, too, begged for an enucleation, which was done on March 30, 1889.

The accompanying drawing (Fig. 12) shows the relative situa-

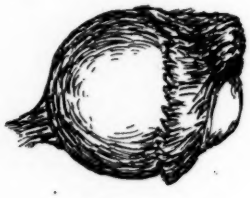


Fig. 12.

tion and size of the growth immediately after enucleation. The microscopical section did not reveal anything essentially different from the preceding case. He was discharged from the hospital

April 5th, and we have heard nothing from him since, as we would have done had there been a return of the trouble in the other eye.

Case XXIV.

DOUBLE SYMMETRICAL UPWARD CORECTOPIA.

Manz in his classical treatise on malformations of the eye in Gräfe and Sämisch's *Handbook*, vol. ii., says that corectopia of high degree in which the pupil is displaced from the centre and drawn far towards the edge of the cornea is rare, and that the displacement is usually downward and inward; and further, that cases of "pure" corectopia in which the iris structure has undergone no changes are very uncommon and of great interest as bearing upon the question of the connection of such displacement with coloboma.

I give the accompanying drawing (Fig. 13) therefore as a

Fig. 13.

representative of such rare conditions. It was in the person of a girl of three years, who was brought to my clinic by her mother, June 26, 1892. The pupil in both eyes was drawn, as shown in the figure, directly upward and reached almost the edge of the cornea. There was, however, a small bridge of iris tissue left at this place. The pupil was unusually small for a child of that age, and was by no means active. The vision seemed to be fairly good. Under atropine, 2 per cent., the pupil dilated to the dotted lines shown in the drawing. The fundus under ophthalmoscopical examination revealed nothing abnormal. There was no coloboma of the choroid, nor any displacement of the lens that could be discovered.

SIMPLE METHOD OF OPERATING FOR PARTIAL TENOTOMY OF THE RECTI MUSCLES (GRADUATED TENOTOMY).

By EUGENE SMITH M.D.,

PROF. OF OPHTHALMOLOGY AND OTOLOGY IN THE DETROIT COLLEGE OF MEDICINE, ETC.

(*With two wood-cuts.*)

SEVERAL years' experience in the following simple manner of making partial tenotomy of the recti muscles in heterophoria, leads me to give it to the profession with the remark that it is as effective as it is easy.

The instruments required are an ordinary eye speculum; a small triangular corneal knife—the stop-keratome of De

Wecker; and a peculiar ring forceps, each branch of which ends in a sharp hook. When closed the forceps form a small ring at the points. (See cuts.)

After cocainization, the eyelids are held open with the speculum, the belly of the muscle is seized at the equator of the eye while the patient looks in the direction opposite to the muscle to be operated on, *i. e.*, inwards if the external rectus is to be cut.

The full width of the muscle and the conjunctiva over it are seized with the forceps, the sharp points of which pierce the conjunctiva and grasping the muscle hold it securely in the ring. The muscle is then drawn away from the eyeball in such a manner as to put the tendon on the stretch, while the point of the triangular knife is pushed through the conjunctiva and centre of the tendon at its attachment to the sclera. If, in the judgment of the operator, the division of the tendon made by the forward movement of the knife is deemed insufficient, or found to be so on testing, the muscle is again seized with the forceps, the knife entered by the same wound in the conjunctiva and tendon, and by lateral movements the tenotomy is enlarged.

The very small wound in the conjunctiva usually closes by its own elasticity. The bleeding or effusion beneath the conjunctiva is very slight, and seldom does over-correction occur.

The pain is slight, and the operation quickly made, a desideratum of more import to the patient than to the operator. No dressing is required.

THE DIAGNOSIS OF SYMPATHETIC OPHTHALMIA.

By Dr. LEOPOLD WEISS,

LECTURER ON OPHTHALMOLOGY, UNIVERSITY OF HEIDELBERG.

Translated by Dr. Casey A. Wood, of Chicago.

At the last meeting of the Ophthalmological Congress at Heidelberg, it was held by several speakers[1] that not infrequently it is difficult to decide whether an inflammation in the second eye, occurring some time after an injury to the first, is or is not a true sympathetic ophthalmia.

It is self-evident that cases of this sort are of great importance to accident insurance companies, because if it be admitted that a wound of one eye has endangered the vision of the other, the injured man would have a just claim for increased compensation.

Having in view the great practical importance of this question, I submit the following cases.

Case 1.—Heinrich E., æt. twenty-eight, boiler-maker, was, Feb. 11, 1887, severely injured in the left eye by a piece of wire. The fragment penetrated the cornea, and passed through the iris into the crystalline. Whether it imbedded itself in the latter, or went through it into the vitreous, it was impossible to say at the time of the first examination, owing to the cloudiness of the lens. Vision was reduced to perception of light.

[1] *Transactions of the Oph. Congress*, pp. 100–113. Schweigger stated that there are no objective signs by which one may recognize the sympathetic character of an irido-choroiditis.

The patient was admitted to hospital, and attempts were made to preserve the eye. During the next few days, however, such marked inflammatory symptoms appeared in the injured organ, and the other one became so sensitive to light when the bandages were changed, that the left eye was enucleated on the 16th of February. Everything progressed favorably, and the patient was discharged February 24th. At this date the right eye was free of irritation, and had normal accommodation and visual acuity. Towards the end of March he was fitted with an artificial eye, but did not return to work until the beginning of October, 1887. During this period he received full benefits; afterwards he was paid one third—the amount allowed for the loss of one eye, the vision of the other being good. E. worked about nine months without complaining of any trouble with his eyes.

At the end of August, 1889, patient returned, saying that for some time past he had not been seeing so well, and that recently it was getting quite noticeable, especially when he had fine work to do. On such occasions he was obliged to strain his eye.

Vision was at this time $\frac{6}{36}$. Pupil reacted promptly to light. The eye was neither markedly painful nor tender, although slightly sensitive to pressure. The ophthalmoscope revealed no gross changes, and yet the uniformly rosy papilla, with its indistinct edges, accompanied by a faint cloudiness of the whole fundus, gave rise to the suspicion of a beginning neuro-retinitis. The stump is not tender on pressure.

The patient was again admitted, and treated by the dark chamber and mercurial inunctions. In consequence of this, the visual acuity at first improved, then it came to a standstill, and later it again declined.

As the disease progressed well-marked alterations showed themselves in the fundus. The disk became paler and paler, and some of the central vessels are edged with white. In the background proper are small bright yellowish-white dots, accompanied by changes in the fundus pigment. In December, 1888, V = $\frac{6}{ix}$ to barely $\frac{6}{xxxvi}$. The field of vision was of almost normal extent, although red and green were not correctly named. Urine free from albumen.

In response to a request from the benefit society for my opinion, I expressed myself as follows: In passing judgment upon this rather obscure matter we are met at the outset by

the query: What causal connection, if any, is there between the injury to the enucleated eye and the serious disease in the other eye which did not set in until a year and a half afterwards? Is the affection of the remaining eye to be regarded as a sympathetic ophthalmia?

Along with this question we may consider whether the early removal of a badly injured eye usually insures the other one against danger of an attack from sympathetic disease. It rarely happens that the second eye becomes affected after enucleation of the injured eye.[1]

If the disease does affect the sound eye shortly after the excision of the injured one, we may feel certain that at the time of the operation the former organ was already beginning to suffer from some very early and easily overlooked symptoms, indicating that migratory disease-bearing elements had already begun to travel from one eye to the other. When, however, the sympathetic disease does not show itself until after a considerable interval the explanation is more difficult.

As is well known, it not infrequently happens that a badly injured eye, after being affected by phthisis bulbi, may remain quiet and free from pain for years; suddenly inflammatory symptoms, more or less severe, set in and the second eye becomes the subject of ophthalmia migratoria. In cases of this sort it is presumed that the causative elements of disease (probably micro-organisms), which have all the time been lying incapsuled (and consequently harmless) within the injured ball have, from some cause or other, been set free. These first produce a new inflammation in the diseased eye, and then wander forth—in all likelihood by way of the lymph channels—towards and finally reach the sound eye, in which they set up sympathetic disease.

One may conclude from this that it is in much the same way that the sound eye is attacked many years after an enucleation of the injured one—the morbific elements had already escaped from the diseased bulb before its removal. The excision does not entirely remove all these germs; some of them

[1] It seldom happens that a prophylactic enucleation gives rise to sympathetic disease in the other eye. Mauthner, *Sympath. Augenleiden*, i., 53.

are left behind, either in the remaining portion of the nerve or in its neighborhood. The inflammatory reaction following the excision fixes them within the orbit so that they cannot escape. Thus they remain for many years encapsuled and harmless, until something occurs to set them free. Then they resume their unopposed and destructive march towards the healthy eye. There is just one weak point in this analogy, viz.: it seems more plausible to suppose that morbific micro-organisms can remain encapsuled and innocuous for years within a phthisical eyeball than in the loose tissue of the orbit.

The first difficulty which the preceding case presents (if we assume that the trouble in the healthy eye is of a sympathetic character) is the tardy appearance of the disease after the enucleation. The second one lies in the local signs. In the majority of instances sympathetic ophthalmia first shows itself as an iritis (or an irido-cyclitis) and finally attacks the whole uveal tract. Less frequently it affects the optic-nerve entrance as a neuritis optica.

If we regard sympathetic ophthalmia as a migratory affection—the morbific elements travelling along the nerve on the diseased side to the chiasma and thence back by way of the other nerve to the sound eye—it is conceivable that the first signs of the disease will manifest themselves at the papilla of the sympathizing eye. In the first part of their journey they will be propagated *in the same direction* as the lymph stream, but in the second stage they must travel *against* the current. There is nothing remarkable about this since micro-organisms in a lymph channel can make progress against the stream.

The above case was one of optic nerve atrophy following a rather marked hyperæmia of the papilla.

Cases of "sympathetic optic atrophy," are described in literature by Mooren, Krenchel, Dransart, and Roosa. Dransart regards the disease as a peculiar form of sympathetic ophthalmia and remarks concerning it that it is often accompanied by other tissue changes, choroidal atrophy, etc., etc.

After experimental demonstrations of the migratory doc-

trine, says Deutschmann, the older idea (that the papillary decoloration is produced by so-called "sympathetic influences") cannot be entertained.

I do not see anything remarkable in the appearances of choroiditic changes since I had an opportunity, some time ago, of seeing Deutschmann's preparations from the animals he had experimented upon. In the neighborhood of the optic-nerve entrance the changes in the choroid were very noticeable.

The peculiarities of the above case, as well as the difficulties in making a diagnosis, incline me to regard it as a rare form of sympathetic ophthalmia.

The insurance company took my view of the situation and gave the injured man an increased annuity.

I shall briefly relate the remainder of the history. The visual acuity gradually diminished until at present the patient can count fingers only when the hand is held close to the eye. The disk is white, the choroidal changes are about the same as before.

I have but a few additional remarks to make. Although I am an adherent of the migration theory, yet I cannot quite agree with the assertion that the optic nerve furnishes the only route over which the morbid elements can travel to reach the sympathizing eye. If this were true it would seem remarkable that in the majority of instances the earliest and most pronounced evidences of the disease should first appear in the anterior and not in the posterior segment of the eye. The statement that the germs commonly enter the second eye at the disk (where they give rise to little or no disturbance), push their way from behind forward (without producing any decided inflammatory changes in their course), and having finally reached the anterior portion of the globe, there for the first time make their destructive influences felt—all this has something problematical about it. As regards the cases of sympathetic neuritis it is next in order to locate the route along which the infection is carried to the optic nerve. Experiments on animals have demonstrated that it can be transmitted in this manner. When the parts in the anterior segment of

the eye are much inflamed, it is difficult, during life, to determine in what percentage of cases the optic-nerve entrance and its vicinity are affected, for even if the ophthalmoscopic examination show a reddened and ill-defined papilla, the latter appearance may be simply an effect produced by hazy media and not pathological changes present in the optic disk.

CASE 2.—The history of the second case will be given only in abstract. J. S., æt. forty-five, was struck in the right eye with a fragment of steel, which entered through the lid into the posterior segment of the ball, causing reduction of vision to mere perception of light. Several months after, the vision of the other eye began to fail. The ophthalmic picture, at first slight congestion and œdema of the retina, developed later into the regular picture of Bright's retinitis. Albuminuria was at first scant and intermittent, later well marked.

A CONVENIENT ARRANGEMENT FOR THE TEMPORARY USE OF PRISMS.

By WILLIAM E. BAXTER, M.D.,

SURGEON TO THE EASTERN MAINE EYE AND EAR INFIRMARY, AND OPHTHALMIC AND AURAL SURGEON TO THE BANGOR GENERAL HOSPITAL, BANGOR, MAINE.

THE prescribing of prisms experimentally for patients is practised frequently by many oculists at the present time, and as many practitioners prefer to furnish these prisms temporarily, one is obliged to provide oneself with a large number of pairs of prisms with the apices set in various directions to meet the demand in all cases. The following arrangement of prisms has been used by me for some time with so much satisfaction, that I trust it may be of value to others to provide themselves with similar sets. The arrangement consists simply of mounting prisms of the same size and shape as the lenses furnished in a Nachet case, in deeply grooved circular frames, and having the screws that retain the lenses a little longer than usual—a thumb-screw is most convenient; by loosening the screw slightly the prisms can be revolved to any required angle. The prisms are marked with a slight scratch showing the direction of the apex, and the frames can be made with various styles of nose-rests, and interpupillary dimensions, and with straight or riding bows. These mounted prisms have been made for me by Queen & Co., Philadelphia.

A REMARKABLE CASE OF COMPLETE PARALYSIS OF THE INTERNUS AND EXTERNUS OF EACH EYE.

By F. C. HOTZ, M.D., Chicago,

PROFESSOR OF OPHTHALMOLOGY, CHICAGO POLICLINIC.

ALTHOUGH in ocular paralysis we are accustomed to meet with all sorts of wonderful combinations of paralysed muscles, the simultaneous paralysis of both interni and externi is certainly a combination we do not often observe. But the following case is still more interesting by its remarkable course and its speedy and complete recovery.

On May 3d Mr. G. F., a German druggist, twenty-two years of age, consulted me in regard to his eye trouble. He has always enjoyed good health except two years ago when he had pneumonia, and five years ago when he was overcome by heat and seriously ill for two weeks; but he recovered completely, and had not felt the slightest after-effect from this heat-stroke all these five years. His habits are regular and temperate; and he is very emphatic in denying the possibility of syphilis, and I have no reason to disbelieve his word.

Now two months ago, while engaged in compounding some medicine, he suddenly experienced a peculiar difficulty in seeing; he found he was obliged to turn his head instead of his eyes if he wished to look from one object before him to another. This visual embarrassment has continued ever since; it is no worse nor any better either; it makes him dizzy in walking, and is attended by a continuous dull headache.

When the patient entered the room my attention was at once attracted by a strange, staring expression of his eyes, which the

examination showed was caused by the patient's inability to turn either eye to the right or to the left. The left eye stood exactly in median position ; the right eye showed a convergence of ten degrees (by the perimeter), but in neither eye the faintest trace of any lateral movement (adduction or abduction) could be observed when the object was carried from the median position to the right or left. When the object was carried upwards or downwards the eyes readily moved up and down, but their movements were strictly limited to the perpendicular line, no adducting or abducting deflexion being possible. Each eye showed normal vision, emmetropic refraction, and read Jäg. 1 up to five inches ; the pupil responded readily to light, and the fundus showed a normal ophthalmoscopic appearance. The diplopia test had a very singular result : in the median line and in left side of the common field of vision, the patient saw two (homonymous) images ; but as soon as the light crossed the median line toward the right the diplopia ceased, and the patient saw one light only in the whole right visual field.

On the 10th of May I exhibited the case before the Ophthalmological Society. The paralysis of both interni and externi was still complete ; but there was also a complete paralysis of the right side of the face. The members present also convinced themselves by repeated tests of the absence of double images in the right field, and were inclined to give a rather unfavorable prognosis.

The next day I received a telephone message that Mr. F. was too ill to go out, and then I heard nothing of him until June 17th, when he walked into the office a perfectly well man ; no trace of facial paralysis, and perfect adductive and abductive movements of each eye ; only the right eye still showed a convergence of 10°. An examination made November 30th showed that this convergence of the right eye had disappeared, and binocular fixation was fully re-established. The perimeter showed the rotation of each eye outwards 65°, and inwards 50°.

As to what happened between May 10th and June 17th, Mr. F. gave the following information : While he was under my observation he had been taking iodide of potassium at the suggestion of another physician, and though it did not agree with him at all (causing dyspepsia and nausea) he persisted in increasing the doses until he took twenty-seven grains three times daily. That was on the day when he appeared before the Ophthalmo-

logical Society; and the next morning he awoke with vomiting blood and all symptoms of a violent gastritis, which his physician at once pronounced as being caused by the excessive use of iodine. He was confined to bed for one week, and during this time the left side of his face also became paralyzed; both arms were paralyzed, and even the legs were affected so much that he was unable to walk alone. After two weeks, when he had recovered from the iodine, he began taking strychnine (gr. $\frac{1}{60}$), and within one week he thought he could notice an improvement. He first recovered the use of his legs and arms, then the facial paralysis disappeared, and finally the eyes recovered their lateral mobility.

A NEW PERIMETER.

By J. EDWARD GILES, M.D.

ASSISTANT SURGEON, MANHATTAN EYE AND EAR HOSPITAL; ATTENDING OPHTHALMOLOGIST, DEMILT DISPENSARY; INSTRUCTOR IN OPHTHALMOLOGY, NEW YORK POLYCLINIC.

(*With a wood-engraving.*)

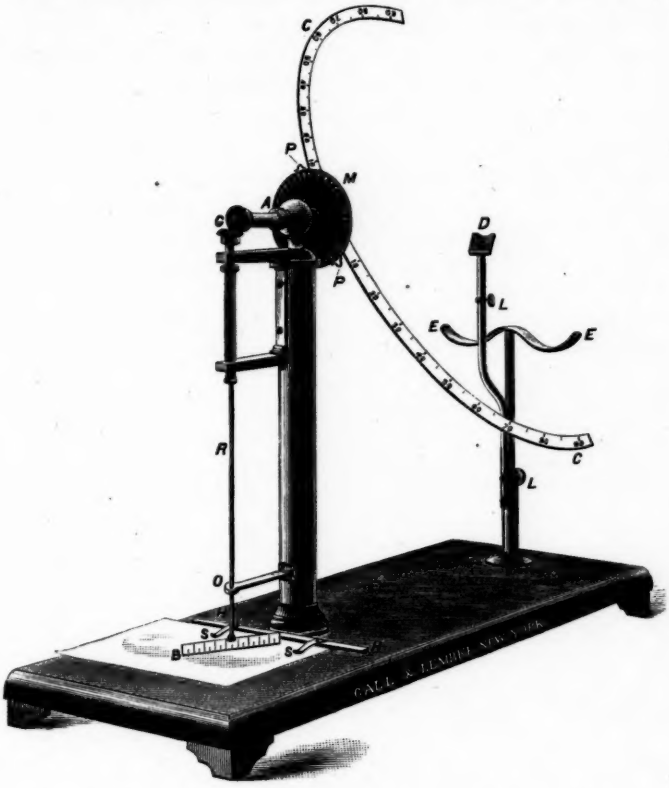
THE perimeter illustrated by the accompanying engraving differs from others in use, chiefly in the device for recording observations, which is seen at the left of the engraving. This device consists of a graduated index-bar *B* fixed at the lower end of a vertical rod *R*, which is connected with the axis *A* of the instrument by means of a bevelled gearing *G* in such a way that every change in the position of the graduated arc *C* is indicated by a corresponding change in the position of the index-bar.

The instrument is designed to be used with Emerson's perimeter charts, but any other chart may be used, or if no chart is at hand, a blank sheet of paper will serve the purpose, because the angle of the graduated arc is always shown by the position of the index-bar, and it is only necessary to mark with a pencil by the side of the index-bar at the degree corresponding with the observed point upon the graduated arc.

If the Emerson chart is used, the chart is to be placed under the index-bar with the base toward the patient and against the brass guide *H*. There it is held by means of the spring clips *S*. In this position every portion of the field as represented upon the chart will be opposite the corresponding portion of the field of the eye under observation, and consequently there will be no danger of recording an

observation upon the wrong side of the chart and thereby reversing the field.

The axis *A* is hollow, so that, if desired, the observer may keep watch of the patient's eye through the opening. At the axis is a graduated circle *M*, the position of which is



fixed, while around it revolve two pointers *P* which are fixed to the arms of the arc.

The readings shown on the graduated circle should correspond exactly with those shown upon the chart by the index-bar. By means of this additional index, the observations, as taken upon the chart, may be verified, and if by

accident the index-bar is moved from its proper position, the true position may always be found by a comparison of the two scales. If the recording apparatus should be broken by any accident, the degrees could be read upon the graduated circle and noted upon the chart in precisely the same way as with perimeters of the ordinary form which are not provided with a recording apparatus.

The other parts of the instrument are easily understood from the engraving. It will be noticed that both the chin-rest *E* and the guide for the position of the eye *D* are adjustable, and are held in any desired position by means of the thumb-screws *L*. It can easily be seen, by looking through the hollow axis *A*, whether the patient's eye is properly centred, because the axis is of such a length that the least deviation from the centre may be noticed.

The engraving was kindly furnished for the purpose of illustration by the makers of the instrument, Messrs. Gall and Lembke, 21 Union Square, New York.

A CASE OF HEMORRHAGIC IRITIS, WITH REMARKS.

By Dr. CHARLES ZIMMERMANN, Milwaukee, Wis.

BEFORE entering into a discussion on the subject I shall give the clinical history of the following case:

A. B., a dredging contractor, aged thirty-three, of healthy appearance, had no constitutional disease, especially no lues nor tuberculosis. His parents are living and healthy. *Nov.* 3, 1888, he came to my office with a recent *iritis* of his *right* eye. Iris discolored, reacting sluggishly to light. A few synechiæ were torn by atropine. He was in bed until *Nov.* 8*th*, when he got up and went into another room of a cooler temperature. But he could not stay up long, on account of cilliary pain setting in soon after this change. In the evening I found the pupil dull and grayish from a very *copious fibrinous effusion into the anterior chamber*. The pupil was round but less dilated. No red reflex upon inspection with the ophthalmoscope. The next morning the exudation had become denser, so that he could not count fingers near by. Inunctions of mercurial ointment were ordered, which favored the absorption strikingly. *Nov.* 10*th*, the effusion was thinner, so that he could count fingers at three feet; *Nov.* 11*th*, a cloudy red reflex could be obtained for the first time on ophthalmoscopic examination; *Nov.* 15*th*, the pupil was free from any deposit; V = fingers in ten feet; he could distinguish the hands of the watch; details of fundus could be seen indistinctly. *Nov.* 19*th*, he left the bed, and *Dec.* 1*st*, the house. *Dec.* 4*th*, V = $\frac{15}{15}$. *The eye recovered perfectly.*

In *January*, 1890, he had a slight *relapse* of *iritis* in the *same eye*. The blue iris discolored to green and ciliary injection, no syn-

echiæ. He was at once put to bed and treated with atropine and diaphoretics, so that he *recovered in a week.*

April 20, 1892, he came again with slight pericorneal injection of the *same (right) eye.* The pupil reacted promptly to light, and as the iris showed no inflammatory symptoms, I attributed the cause of redness to some dust in his conjunctival sac, which I removed, and gave him cocaine as an antiphlogistic. *April 21st,* status idem. In the evening he went to a wedding party and danced considerably until the early morning. The consequence was a pronounced *iritis of the right eye.* An adhesion at the upper periphery became apparent after dilatation of the pupil by atropine. In the afternoon it was torn, iris green; the whole front of the eye intensely red; no pain. *April 24th,* put to bed, as a *gray exudation in the pupillary area* had formed. *April 25th,* the effusion being larger and chemosis having set in, six leeches were applied to the temple at 6 P.M.

April 26th: more than the lower two-thirds of the anterior chamber were filled with dark coagulated blood.

Only the upper portion of iris was visible. It showed intense swelling, was very much congested, and looked dull from fibrinous effusion into its structure. I ordered inunctions of mercurial ointment and diaphoretics. *April 27th,* the amount of blood had somewhat diminished. Pupil still covered by fibrinous deposits, which had also become less, so that the upper periphery of the pupil gave a red reflex upon ophthalmoscopic examination. $V =$ 0. *April 28th,* he took salicylate of soda 5,00, every hour 1,00, with remarkably good result. *April 29th,* the largest portion of the pupil was free, and on *May 3d,* after repeated doses of salicylate of soda, there was no longer any trace of blood or fibrine in the anterior chamber.

He recovered very nicely. June 1st, V in that eye was $\frac{15}{16}$. During this last attack and even after convalescence, until July, he felt some pain in the muscles of his left upper arm, which appeared to be rheumatic. The patient is very much exposed to the injurious action of dampness, wind, and cold, being engaged in dredging rivers and lakes and building foundations in swamps, which may account for the frequent relapses of iritis.

The most striking feature in this case is the association of iritis with hyphæma, i. e., its hemorrhagic character.—The occurrence of hemorrhages during the course of iritis is

regarded as extremely rare, "exceptional" according to Wecker,[1] and, as far as I was able to peruse the incident literature, only a few publications exist on it by Hutchinson,[2] Keyser,[3] Reche.[4] In order to find an explanation for this complication, it be may well to consider *what part the blood plays in iritis.*

1. One of the first and usual signs of iritis is *hyperæmia.* Not only are the extremely numerous blood-vessels of the iris, which form the most prominent streaks of its surface, extended and injected so that they appear as red loops and nodules, but even a number of smaller vessels, not visible before, become quite pronounced now by their engorged condition. Wecker (*l. c.*) believes in a new formation of blood-vessels on the surface of the iris, and thinks that the so-called ecchymoses of the iris, if carefully examined through a magnifying glass, may be found to be mostly convolutions of small blood-vessels.

2. If we go a step further, some of the *coloring matter* of the *blood* will *transude* through the walls of the blood-vessels of the iris, which are pretty thick on account of their well-developed muscular structure, imparting a different tint to the natural color of the iris by *infiltration of its stroma with hæmoglobine.*

3. A higher degree of inflammatory reaction will be indicated, if the *walls of the blood-vessels* undergo such a change, that they *become permeable for* the *red-blood corpuscles*, which pass them and enter the tissue of the iris. Then we have extravasations of blood into the substance of the iris. Larger hemorrhages especially happen between the posterior limiting membrane and the pigment layer.[5] The swelling and infiltration of the structure of the iris are partly due to them. On the surface of the iris we see such hemorrhages in gummatous or condylomatous iritis, in which the yellow nodules are sometimes streaked with blood.[6]

[1] Graefe-Saemisch, iv., p. 492.
[2] *Ophth. Hosp. Rep.*, 1874, viii., p.3, and *Nagel's Jahresbericht*, v., p. 327.
[3] *Philad. Med.* and *Surg. Rep.*, 1874, p. 211, and *Nagel's Jahresber.*, v., 323.
[4] *Klin. Monatsbl. f. Aughenh.*, 1892, May, p. 176.
[5] Michel, *Lehrbuch der Augenh.*, ii. Aufl. p. 313.
[6] Noyes, text-book on *Diseases of the Eye*, 1890, p. 408.

4. There will be severer varieties of iritis, in which the *red blood corpuscles* not only penetrate through the walls of the vessels, but even through the tissue of the iris and *form a part of the effusion into the aqueous humor.* In fibrino-plastic iritis, *e. g.* the coagulating matter of the aqueous is increased partly by the presence of lymphoid and pus cells and red blood corpuscles, causing the turbidity of the aqueous. Red blood corpuscles are also found in the fibrino-purulent forms of iritis and very often in the accompanying hypopyon, which shows sometimes streaks of blood; or its color becomes changed at the bottom from an accumulation of red blood corpuscles. This is even more marked in *spongy iritis* (Knapp), according to the examinations of Arlt, who "described the exudation as sero-fibrinous and hemorrhagic, in which the fluid and cell elements tend to separate."[1]

5. The most pronounced appearance of blood, of course, occurs when the *red blood corpuscles* escape in such masses as to *fill the anterior chamber* partly or wholly and form a *regular hyphæma.* Most authors saw this in purulent iritis. Hyphæma alone (without hypopyon) as a complication of iritis is mentioned as a rare phenomenon by Klein,[2] Vossius,[3] Keyser,[4] and Reche.[5] Hutchinson[6] noticed hemorrhagic iritis, (1) after operations; (2) in sympathetic iritis, in which hyphæma is a very unfavorable symptom; (3) in chronic iritis of old people; (4) in certain arthritic individuals.

If we ask for the *causes* of the *spontaneous hemorrhages of the iris* in general (not considering the traumatic), we may look for them under two different headings:

1. *Hemorrhages,* dependent upon *diseases of the blood-vessels,* as atheroma or fatty degeneration. The latter condition very likely prevails, when violent efforts in spells of vomiting, coughing, sneezing, or during labor, in pulmonary emphysema, or heart diseases lead to hemorrhages into the anterior chamber (the increased pressure of expiration augmenting the tension in the veins and capillaries and

[1] Noyes, *l. c.*
[2] Klein, *Eulenburg's Real-Encycl.*, Aufl. ii., vol. viii., p. 624.
[3] Vossius, *Grundriss der Augenh.*, 1888, p. 200.
[4] Keyser, *l. c.* [5] Reche, *l. c.* [6] Hutchinson, *l. c.*

causing ruptures of their walls). The hemorrhages observed in vascular or teleangiectatic[1] growths,[2] *e.g.* in granulomata, in gummata, or condylomata, are a consequence of the degeneration of the wall of the vessels enclosed in the neoplasma. Some general affections leading to hemorrhages anywhere in the body may cause hyphæma, *e.g.* scurvy,[3] pernicious anæmia, anæmia from great losses of blood.

2. *Hemorrhages from sudden changes of the intraocular pressure.*[4] In plastic irido-choroiditis with complete occlusion of the pupil, the increased tension may suddenly fall to a minus condition and give rise to an extravasation of blood into the anterior chamber. The same is known of eyes which have become phthisical in consequence of cyclitis. Wecker (*l. c.*) saw hyphæma in cases of absolute glaucoma or in earlier stages of glaucoma, if its hemorrhagic character was indicated by multiple retinal hemorrhages, or in the glaucomatous period of intraocular tumors. The sudden decrease of intraocular pressure during the performance of iridectomy in glaucoma produces sometimes copious hemorrhages from the iris.

In our case I noticed the hyphæma at 8 A.M., at the first visit after the leeches were applied (at 6 P.M. on the previous day), *i.e.*, after about fourteen hours, no observation being made in the meantime, so that it may have occurred sooner. In that event we might assume that the intraocular pressure, which is often augmented in iritis,[5] suffered an instantaneous diminution by the depletory effect of the leeches and gave rise to the bleeding.

According to Arlt,[6] "a collection of blood in the anterior chamber may in many cases be referred to *inflammatory changes in the ciliary body* and thereby impeded reflux of blood to the venæ vorticosæ." This however was not here the case, as the chief diagnostic symptoms of cyclitis were wanting. There were no pathological changes of the deeper

[1] Schelske, *Lehrb. d. Aug.*, 1870, pp. 72–97, and *Nagel's Jahresber.*, i., p. 298.
[2] Knapp, intraocular tumors.
[3] Michel, *l. c.*, p. 319.
[4] Wecker, *l. c.*, p. 573.
[5] Schweigger, these ARCHIVES, xx., p. 477.
[6] Arlt, *Clinical Studies on Diseases of the Eye*, translated by L. Ware, 1885, p. 279.

uveal structures, no manifest deviation from normal tension, and especially no pain or even tenderness on gentle pressure on the upper portion of the ciliary region.

In the three cases of Reche (*l. c.*) a *rheumatic disposition* existed, which he considered as the *etiological element* of the *hemorrhagic iritis*, but in what manner he does not say. On the other hand Noyes[1] asserts that "rheumatic and gouty iritis are more *serous*." But as it is claimed that the gouty or rheumatic, or syphilitic dyscrasia are frequent factors in retinal hemorrhages from a weakened condition of the vessels,[2] they might also lead to hemorrhages of the iris, in spite of the thicker walls of its vessels in comparison to those of the retina. And indeed Hutchinson (*l. c.*) saw in certain arthritic individuals each attack of iritis commence with a hemorrhage into the anterior chamber.

This, however, does not apply to our patient, who, being otherwise perfectly healthy cannot be said to have suffered from any dyscrasia, even if he had some rheumatic pain in his arm.

The iritis itself being very violent, since it attained such a high degree in a very short time, it seems to me that its *hemorrhagic character* was not dependent upon any special etiological type, but that it was simply a *sign of its intensity* from the following reason: The red blood corpuscles permeate the walls of the capillary vessels by diapedesis, and will be the more numerous the more the capillary network of an organ is developed. *An effusion becomes chiefly hemorrhagic if the walls of the capillary vessels are very much changed by the inflammation; and consequently their circulation is considerably impeded, as it happens in* VERY SEVERE INFLAMMATIONS.[3]

This general law gives the most satisfactory explanation of our case, which shows, that *iritis of very intense character may now and then be complicated by hemorrhages.*

---

[1] *L. c.*, p. 410. [2] *Ibid.*, p. 553.
[3] Cohnheim, *Vorlesungen über allgemeine Pathologie*, i., p. 217.

HISTORY AND PRINCIPLES OF KERATOMETRY; ITS VALUE AND LIMITATION IN THE CORRECTION OF ASTIGMATISM.

By CARL WEILAND, M.D.,

ASSISTANT IN THE EYE DEPARTMENT OF JEFFERSON MEDICAL COLLEGE HOSPITAL OF PHILADELPHIA.

(*With five drawings.*)

THOMAS YOUNG, the celebrated natural philosopher, was the first who in 1793 discovered the astigmatism of the dioptric system of the human eye. He observed it in his own person, where it amounted to about 1.5 D, and concluded from different experiments that it had its cause in an oblique position of the crystalline lens. The second known case of astigmatism is that of the English astronomer Airy, who in 1827 described his own case, but, in the absence of any satisfactory reasons, wisely refrained from expressing any opinion as to the seat of his defect; nor does it seem that he tried any experiments to elucidate that point. Other cases were reported by Dr. Goode in 1848. Here the lens was suspected, for he states expressly that, in a case of high astigmatism, he in vain endeavored to find by the form of a reflected image any peculiar asymmetry of the cornea and that he therefore was inclined to look for the source of the trouble in the crystalline lens. The first, however, who found the cause of the astigmatism in the cornea, was Dr. Thompson at about the same time, though it is not said by what method the result was obtained. Later, in 1855, Wharton Jones and Wilde simply assert that the cornea in its vertical meridian has a shorter radius of curvature than in the horizontal one. But they did never observe it themselves, and, as up to their time no exact measurements of the cornea had been performed, except

one, it seems rather probable that these authors simply ventured upon that assertion without a scientific basis for it. They hardly deserve credit therefore for that statement; but they may serve, as Donders sarcastically remarks, to show that sometimes even in science we may observe the truth of the old words: *Audaces fortuna juvat.*

It was only after exact measurements of the cornea had been made, that the question about the action of the cornea in astigmatism could be settled. And it was not long till they were made. It is true that already more than one hundred years before, in 1723, the Frenchman Petit determined the curvature of the cornea in dead animals by cutting segments of different circles into pieces of copper, and then trying which of these segments exactly fitted the cornea; by which means he obtained pretty accurate results, and first observed the flattening of the cornea near the limbus. Thomas Young also, in 1801, had determined in a very ingenious way the curvature of his own corneæ, and in 1832 by C. Krause and later, in 1847, by C. Brücke elaborate measurements of the cornea had been made, but simply by compasses and finely divided rulers. But these were all not exact enough for the purposes of physiological optics, where accuracy down to $\frac{1}{100}$ of a millimetre is demanded. A better principle had to be applied, and that was found in the study of the images of objects reflected from the cornea. For the anterior surface of the cornea is a little convex mirror, and the known laws of catoptrics allow us to find the radius (r) of a spherical, convex-reflecting surface if the size of the object (O), the size of the image of this object on the mirror (cornea) (I), and the distance of the object from the mirror (cornea) (d) are known. These four quantities are in the following relation $\frac{\text{Object}}{\text{Image}} = \frac{O}{I} = \frac{2d + r}{r}$, from which is found $r = \frac{2dI}{O - I}$.

Now in this expression for r, the quantities d and O can be easily and accurately measured, and all that remains still to be put into this formula is the value for I, the size of the corneal image. If this could also be easily measured, r could be found quickly by a simple calculation. *The whole*

problem, therefore, reduces itself to the measurement of this small corneal image. This problem is the same as that of measuring the size of small heavenly bodies, or the angular distance between two stars in astronomy; so that almost the same instruments might be used, if only the astronomical telescope of long focus be changed into one of short focus. And, indeed, the science of measuring this small image on the cornea, ***keratometry***, has passed through similar phases as micrometry in astronomy. The first instrument used in astronomy was a filar micrometer, in which two fine threads of a spider's web are movably so arranged that each can be made to coincide with one end of the small object. From the observed distance between these two threads, then, the size of the observed object, or the angular distance between two stars, is easily calculated. And this principle was first employed for the cornea by R. H. Kohlrausch, in 1840, and Senff, in 1846, the latter of whom even succeeded in measuring the horizontal and vertical radius of curvature of the cornea, and so finding the ellipticity of its form. But this method is not a good one for the human cornea, for the reason that while the heavenly bodies are quiet and do not oscillate during the observation, the eye almost constantly performs little jerky movements, so that it is very hard, if not impossible, to make the two threads coincide with the two end-points of the image at the same time; as any one, who has a microscope, can form a good idea of, if he will simply try to measure a small moving body on the stage with an eye-piece micrometer.

This method, therefore, was given up soon and another employed, long before used in astronomy, and first introduced into that science by Savary in 1743. It consists in doubling the little object looked at, and bringing these two objects in such a relation to each other that they are just in contact. Suppose that the object looked at was

Fig. 1

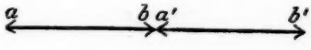


Fig. 2

ab (Fig. 1), and that we had some means by which we could not only leave *ab* there, but at the same time displace each point of it a certain distance. Then we would get two objects, and if we made the displacement of each point equal to *ab*, it is evident that a^1, the displaced *a*, would fall upon or just touch *b*, and that we would get a configuration like Fig. 2. We observe now that, if the two images touch each other, the displacement of each point must be equal to the size of the primary image, and that we shall know the size of this image, if we can find the displacement of two homonymous points when there is contact between the two images. We measure, then, the size of the corneal image by the displacement that must be given to each point, in order to produce contact between the two images. This principle is a great improvement, because if now the observed eye moves a little, both images move in the same way, so that it is still very easy to produce contact.

Savary's method, as modified by Dollond in 1754, and still almost exclusively used in astronomy, consists in cutting through an achromatic objective directly through its optical centre, and then framing these two halves so that they can be slided upon each other, thereby producing at the same time the effect of a prism and of two equal lenses, each of which gives rise to one image. The size of the observed object (*I*), or in our case, the dimension of the corneal image (*I*), the focal distance of the objective used (*f*), the distance of the corneal image from this objective (*d*), and the displacement between the two centres of the semi-lenses (*e*), necessary to produce contact between the two images, are in the following relation to each other: $I = \frac{e\,d}{f}$.

Now as *d*, the distance of *I* from the objective, and *f*, the focal distance of this objective, can be readily determined, it is only necessary to find *e* in order to obtain *I*. If we therefore measure the value of *e* by a fine screw, as is done in astronomy, we have all the data for calculating *I*. But, as the determination of *e* requires a very costly mechanical arrangement, it is more convenient for our purpose to once for all fix *e*, the intercentric distance, so that together with

given values of d and f, a certain value of I, say 3 *mm*, is produced. And if further we made $d = 2f$, that is, twice the focal distance of the objective, then we would have $I = \frac{e\,2f}{f} = 2e$. Now it is well known that if an object, here the corneal image, is at $2f$ from the objective, then the image of this by the lens will appear at $2f$ behind the lens and be of exactly the same size as the real corneal image, a fact also employed in Javal's instrument. If, therefore, the corneal image is 3 *mm*, we have to make $e = 1.5$ *mm*; then there will appear at $2f$, behind the lens, 2 corneal images, each of the same size as that on the cornea = 3 *mm*, and therefore just touching each other, provided the cornea is $2f$ *mm* from the objective. Now in our formula for $r = \frac{2dI}{O-I}$ the values for d and I are constants by our arrangement, and we only have to observe for each case the value of O, or the size of the object necessary to produce at $2f$ from the cornea a corneal image $= I = 3$ *mm*. This can be easily done by moving one end-point of O till the two corneal images are just in contact, as will be described more in detail later on. An instrument of this description for keratometry was proposed by Tscherning in 1887, and I know by actual experiment that it can be easily constructed. If we took *e. g.* an achromatic lens of + 4. *D*, the distance between the centres of the two halves would have to be 1.5 *mm* in order to produce from a corneal image of 3 *mm* at the distance of 500 *mm* from the eye, two corneal images exactly in contact and each equal = 3 *mm*, at 500 *mm* behind the objective, where it could be conveniently enlarged by an eye-piece.

This modification of an old astronomical method, though it seems so natural to think of it, was not made use of then in the history of keratometry. It was another later device, also by an astronomer, that gave in a great man's mind the impulse to the first celebrated ophthalmometer. In 1841 the astronomer Clausen first proposed to use two plane glass-plates in order to get the double images of the stars. One of his plates was stationary, while the other was movable; so that the rays of light from an object in the axis of the telescope

would fall perpendicularly upon one plate, but obliquely upon the other. Now it is a fact that, if we look through a plate of glass of the thickness δ at an object lying in a line at right angles to the plate, we see the object in its old place, but we see it displaced laterally as soon as the plate is turned by an angle α, which displacement is $= \frac{\delta \sin(\alpha - \beta)}{\cos \beta}$ where δ is the thickness of the glass, α the angle of rotation of the plate, and β is an angle found by the well known law, $\sin \alpha = n \sin \beta$, if n is the index of refraction of the glass used. This device was perfected by Helmholtz. He made both plates rotate equally, and putting them in a box in front of a telescope for short distances, he so constructed his ophthalmometer, with which up to about a decade ago all the important work in physiological dioptrics has been done. In the instrument as made by Meyerstein in Göttingen, and which was used by the author for some time,[1] there was $\delta = 4.3972$ *mm*, and $n = 1.518$, so that for a corneal image of 2 *mm* the rotation of each plate had to be 33° 42′. For a corneal image of 2.1008 *mm* α was = 34° 57′, which shows how sensitive the instrument is, especially as it allows to read off the tenths of a degree.

After Helmholtz in 1854 had published his method and results about the exact form of the cornea, he was soon followed by men like H. Knapp, F. C. Donders, and others. By their labors the physiological and pathological shape of the cornea in all meridians was investigated, so that very little was left to be added to their work. They found first that the cornea is the main but not the only seat of the astigmatism. But to that question we shall return later on. First we must add a few more remarks to our historical sketch. The instrument of Helmholtz is used in such a manner that the object reflected from the cornea remains unchanged. Here the object is the imaginary line between two points of light, *a* and *b*.[2] These are reflected from the cornea,

[1] The privilege of its use for some months was kindly granted to me by its owner, Dr. Geo. Strawbridge of Philadelphia, to whom I express herewith my sincere thanks.

[2] I know very well that not two, but three lights or mirrors are used, but as the principle involved is the same as with two, the simpler arrangement is chosen for the description of the principle.

and if the second image of a, that is a^1, does not fall on b, the glass-plates are turned till they do so. Then the displacement equals the size of the corneal image $ab = \frac{2\delta \sin(\alpha - \beta)}{\cos \beta}$ where δ α, and β have the meaning before mentioned. Middelburg added to the armamentarium a large copper ring, also used by Donders, on which he moved the line of his lights by the same angle as the box of the ophthalmometer, so that in that way he was able to measure the radius of curvature of the cornea in all possible meridians.

Although this instrument of Helmholtz has done so much for scientific research, it has some disadvantages which do not allow of its being used very well outside of a laboratory. The instrument must be off from the observed eye at least six feet, the box with the glass-plates must be turned separately from the lights around the axis of the telescope, and, what is the greatest objection to its clinical use, for the determination of each radius not less than eight careful readings of a fine nonius have to be made, *while not less than sixteen readings are required for finding the corneal astigmation.* It takes therefore a great deal of time and labor and so has never come into general use. Something simpler was wanted for clinical purposes. Here the first thing naturally was to simplify Helmholtz's instrument, and this was first done by Javal and Schiötz in 1880, who, however, afterwards left this principle for another one. Another modification that is used by the author, is the modification by Leroy and Dubois. It has the two glass-plates, fixed at a certain angle to each other, thereby producing a certain fixed displacement of about 3 *mm.* The contact of the double images is regulated by sliding on a graduated *straight* bar a reflector, which forms one end of the imaginary line, the object, while the other end is represented by a stationary reflector. In its present condition the instrument requires careful manipulation and a very good light to give accurate results, but, with some improvements and the addition of a headrest, may well compete with Javal's, especially as it can be made at less cost, but it must be admitted that for the determination of the meridian it is inferior to Javal's.

However, it was not this principle which was simplified

first, but an entirely different one. Coccius was it, who, in 1867, first employed that new principle of producing the double images. This principle had also been employed before in astronomy, having been invented and first applied there by Rochon, in 1801. In this method a prism or rather two prisms of quartz, a double-refracting substance, are used, which, by arranging all the rays from an object into two differently refracted sets, an ordinary and an extraordinary one, make thus the object appear double.[1] The amount of displacement it produces depends with a given prism on its position in the telescope, and for that reason it is given a fixed position in the tube in such a manner that, under the conditions used, a certain displacement, say 3 *mm*, is obtained. Then we have in our formula for $r = \frac{2\,d\,I}{O-I}$, the values d and I always the same, and we have to arrange the object so that the two images of it on the cornea are exactly in contact. This is, in the main, the principle of the latest instrument, that at present plays such an important part in the discussions of the ophthalmological societies.[2] Javal's ophthalmometer indeed is at the present time the best instrument that we possess to quickly determine the form of the cornea, and it ought to be used more extensively by the profession. It consists in the main of a double-refracting prism, which is placed in a telescope so, that at twice the focal distance of the objective used, which distance is marked in the tube by two fine, crossed threads, there appear of each point on the cornea two points, 3 *mm* apart. Should, therefore, the little image on the cornea be 3 *mm* long, then the double images will just touch each other. Javal uses as his object an imaginary line, the dis-

[1] Different authors, among them even Noyes, speak of this prism as a Nichol's prism. This is a mistake, as a Nichol's prism never produces double images of an object, but is rather used to suppress one, the ordinary one.

[2] There have been several attempts to replace the glass-plates and double-refracting prisms by two simple prisms of crown glass, *e.g.*, one by Landolt, in 1878, who so constructed his diplomètre. Besides Schöler and Mandelstamm (1872) invented their micro-optometer and Donders (1872) his corneal microscope for ophthalmometric measurements. But these attempts have not been very successful. One new method, however, has been employed by Blix, of Sweden, in 1880, by which he can measure the radius of curvature at one point of the reflecting surface (*Oftalmometriska Studier*, Upsala, 1880). But space forbids a description of this ingenious plan.

tance between the inner edges of two white reflectors *A* and *B*; one of which *A* is a full rectangular figure, while the other *B* is divided into steps. These reflectors slide on an arc[1], that is so fixed to the tube of the telescope that it can be only rotated together with the tube and the prism around the axis of the instrument. Suppose, for example, that the two reflectors have been so arranged that A^1 and *B* just touch as Figure 3 shows.

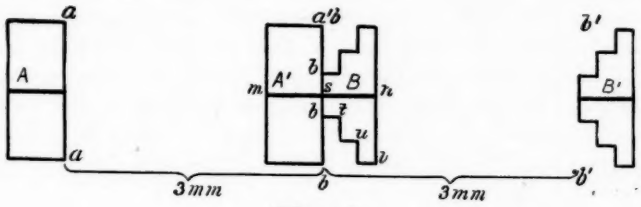


Fig. 3

Here *A* and A^1 are the double images of one reflector, and *B* and B^1 those of the graded one. The distances $a\,a^1$, and $b\,b^1$ are each = 3 *mm*; they represent the displacement of homonymous points, while *ab* is the corneal image to be measured and which here is = $a\,a^1$ = 3 *mm*. We also see here that the two black lines on reflector A^1 and *B* are in one straight line (*msn*), which position must be always sought for, as it indicates that we are in a main meridian. Let us now turn our instrument around its axis, till we again come to a position where the two black lines *ms* and *sn* are in one straight line *mn*; then if the radius of curvature is the same here, we will, of course, see also no change here in our corneal double images; but if the radius be smaller, we will observe that *B* partially overlaps A^1. This must be so, for as $r = \frac{2\,d\,I}{O-I}$ we must, *d* and *O* having remained the same, get a smaller *I* for a smaller *r*. Therefore, our corneal image of *O* must become smaller than before, smaller than *ab*; while $a\,a^1$

[1] This ought to be a straight line, because on an arc the distance *d* of object and cornea in continually changing, just as the distance of a chord from the centre of a circle is changing as long as the chord changes.

and $b\ b^1$, the displacements of homonymous points, remain still the same by virtue of the prism. If then the distance between A and A^1 and that of B and B^1 remain the same, while the distance between A and B gets less, it is evident that B must fall on A^1; partially or wholly, that depending on the smallness of the new corneal image. Suppose, then, that now B overlapped A^1 as far as u or two steps, we would then know at once that the new O, call it O^1, would have to be two steps larger in order to have a corneal image $= a\,a^1$ or 3 *mm*, as before in the first meridian. Now the question arises, how much larger do we have to make our object, or how large our steps, that each shall indicate just one dioptry increase of refractive power or corneal astigmatism. To answer this we must remember that the *posterior* focal distance of the cornea is $F_1 = \frac{rn}{n-1}$ if r is the radius of curvature, and $n = 1.3365$, the refractive index of cornea and aqueous humor. Now we always express the refractive power by dividing 1000 by the focal distance, the latter expressed in *mm*. We must divide 1000 by the *posterior* focal distance, as only the *posterior* focal distance of the cornea is an indicator for the refractive power of the cornea for rays that come from the outer world. It is therefore a mistake if Javal takes the anterior focal distance which is $\frac{r}{n-1}$. But more of that later on. We have, then, for the refractive power of the cornea D the value $\frac{1000}{F_1}$, so that we get $D = \frac{1000}{F_1} = \frac{1000\,(n-1)}{rn}$ and if we substitute the value for $r = \frac{2dI}{O-I}$ we have $D = \frac{1000\,(n-1)\,(O-I)}{2dnI}$. Now suppose that in another meridian of smaller r we have to make $O = O^1$ in order to get contact, or at least to get a corneal image of the same size as before, then we have here $D^1 = \frac{1000\,(n-1)\,(O^1-I)}{2dnI}$. The difference in refractive power between the two meridians will then be $D^1 - D$, which $= \frac{1000\,(n-1)\,(O^1-O)}{2dnI}$. Now in Javal's instrument the distance (d) of the object from the cornea is 281 *mm*, the image I always

$= 3$ *mm*, and as $n = 1.3365$ we have $D^1 - D = \frac{336.5\,(O^1 - O)}{2n\ 281 \times 3}$ $= \frac{O^1 - O}{6.7}$; that is, if $D^1 - D = 1$, then $O^1 - O = 6.7$ *mm*, or we have to make the object 6.7 *mm* larger for each dioptry increase of refractive power. Each step would, therefore, have to be 6.7 *mm* large; but Javal makes his steps only 5 *mm* for each dioptry of astigmatism. What is the cause of that? The cause for this discrepancy lies in the *different meaning of the term corneal astigmatism.* We meant with it so far the difference of the actual refractive powers of two main meridians, $D^1 - D$, *while Javal means by that term the cylinder, which corrects this* $D^1 - D$. This is by no means the same as we shall see in a moment.

To elucidate this point and several others about the correction of the real corneal astigmatism $D^1 - D$ by cylinders, and also to show why Javal makes his steps each $= 5$ *mm*, we put the general question in the following way. If there is given an astigmatic cornea, which has in one main meridian the refractive power D, while in the second main meridian at right angles to the first it has the refractive power of D^1 dioptries, what is the glass that has to be placed at the distance d from the cornea in order to correct the astigmatism? But as generally there will be also axial ametropia, the question must rather be brought into the following form. What are the two cylinders which, at the distance d from the cornea, will bring the rays of distant objects to a focus on the retina in each main meridian? For if we have corrected the two main meridians, our problem is apparently solved. This question, as far as I know, has never been put in this general way before, but it must be done; for it will be shown that the former proposition, simply to find the cylinder that will bring the rays of light through both main meridians to the same focus behind the cornea, after they only passed through cornea and aqueous humor, will not be sufficient, if we put the glass at the distance d from the cornea. In order to prove that, we have to recall for a moment a few formulæ about the dioptrics of the human eye. The accompanying diagram will help us to see the space-relations of the different points.

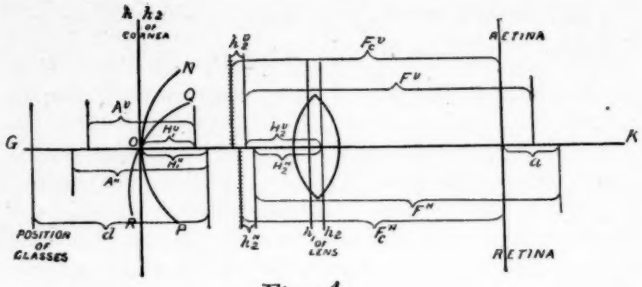


Fig. 4

Here *NOP* is the vertical meridian, or that of smallest r. *QOR* is the horizontal meridian, or that of greatest r. The retina and lens will be recognized at once, though the lens has been removed too far from the cornea for the sake of clearness. All the points, lines, and letters above the optical axis *GK* belong to the vertical, while those below *GK* belong to the horizontal meridian. In the horizontal meridian we have for the anterior focal distance of cornea $A_1^{H} = \frac{r}{n-1}$ if r and n have the meaning formerly given. We also know that the posterior focal distance of cornea $F_1^{H} = \frac{rn}{n-1}$ and as we call $\frac{1000}{F_1^{H}}$ the refractive power of the cornea in the horizontal meridian $= D^{H}$ we have $F_1^{H} = \frac{1000}{D^{H}}$ and therefore we have $A_1^{H} = \frac{1000}{nD^{H}}$. Let now the crystalline lens suspended in aqueous and vitreous humor have a focal distance of F_2 and let it be δ *mm* away from the apex of the cornea, then we find by the use of Helmholtz's well known formulæ for uniting two optical systems with known cardinal points into one: that the first principal point H_1^{H} of the eye, or the combined system of cornea and lens in the horizontal meridian, lies at $H_1^{H} = \frac{\delta A_1^{H}}{F_2 + F_1^{H} - \delta}$ behind the cornea. This by our former values for A_1^{H} and F_1^{H} becomes $H_1^{H} = \frac{1000\,\delta}{n(1000 + (F_2 - \delta)D^{H})}$. By the same formulæ we find for the distance of the second principal point of the eye from the second principal point of lens

(h_2) the value $H_2^{H} = \frac{\delta F_2 D^{H}}{1000 + (F_2 - \delta) D^{H}}$. In the same way we obtain for the corresponding values in the vertical meridian $H_1^{V} = \frac{1000\,\delta}{n(1000 + (F_2 - \delta)D^{V})}$ and

$H_2^{V} = \frac{\delta F_2 D^{V}}{1000 + (F_2 - \delta) D^{V}}$, where $D^{V} = \frac{1000}{F_1^{V}}$ is the refractive power of the cornea in the vertical meridian, and the other quantities are the same as before. Again we find by the same theorem the distance of the anterior focal point of the eye from the first principal point H_1^{H}, which we call A^{H} the value $A^{H} = \frac{F_2 A_1^{H}}{F_1^{H} + F_2 - \delta}$ which is $= \frac{1000\,F_2}{n(1000 + (F_2 - \delta)D^{H})}$. We also find $A^{V} = \frac{1000\,F_2}{n(1000 + (F_2 - \delta)D^{V})}$. But it must be remembered that this A^{V} is reckoned from H_1^{V} as the figure (4) indicates. The distance F^{H} of the posterior focal point of eye from H_2^{H} is $F^{H} = \frac{1000\,F_2}{1000 + (F_2 - \delta) D^{H}}$, and the same distance in the vertical meridian from H_2^{V} is $F^{V} = \frac{1000\,F_2}{1000 + (F_2 - \delta) D^{V}}$.

Let us now suppose that both our cylinders are placed at the distance d from the first principal point of the horizontal meridian or at $d - H_1^{H}$ from the cornea. Then we have for the combined system of cylinder, with the focal distance f, and horizontal meridian of the eye, the following formulæ:

$$h_1^{H} = \frac{df}{d - A^{H} - f} \qquad A_C^{H} = \frac{A^{H} f}{A^{H} + f - d}$$

$$h_2^{H} = \frac{dF^{H}}{d - A^{H} - f} \qquad F_C^{H} = \frac{F^{H} f}{A^{H} + f - d}$$

Here A_C^{H} means the anterior focal distance of the *combined* system of cylinder and eye in the horizontal meridian, and is reckoned from h_1^{H}, which itself is reckoned from the centre of the cylindrical lens. The other quantities have corresponding values. The same formulæ we can write down for the vertical meridian, only remembering that the cylinder has now the focal distance f_1, and that it is placed not at d from H_1^{V}, but at $d - (H_1^{H} - H_1^{V})$ from it. Then we have

$$h_1^{V} = \frac{[d - (H_1^{H} - H_1^{V})]\, f^{1}}{d - (H_1^{H} - H_1^{V}) - A^{V} - f^{1}} \qquad A_C^{V} = \frac{A^{V} f^{1}}{A^{V} + f^{1} - \{d - (H_1^{H} - H_1^{V})\}}$$

$$h_2^{V} = \frac{[d - (H_1^{H} - H_1^{V})]\, F^{V}}{d - (H_1^{H} - H_1^{V}) - A^{V} - f^{1}} \qquad F_C^{V} = \frac{F^{V} f_1}{A^{V} + f^{1} - \{d - (H_1^{H} - H_1^{V})\}}$$

Now to find the value of f in the horizontal meridian it is clear by the diagram that the following equation must be satisfied, if the cylinder f shall bring the rays to a focus at the retina. $h_2^H + F^H = F_C^H + a$. Here a means the axial deviation of the eye, which in our figure is supposed to be hypermetropic. From this equation we have, after substituting the values before found, $a = \frac{F^H A^H}{A^H + f - d}$ If we now call the cylinder that corrects the horizontal meridian Δ, expressed in dioptries, and which we must find by actual trial, then, as of course $\Delta = \frac{1000}{f}$, we get $a = \frac{\Delta F^H A^H}{1000 + (A^H - d)\Delta}$ (I)

or if we substitute the values for F^H and A then

$$a = \frac{\Delta\, 1000^2 F_2^2}{(1000 + (F_2 - \delta) D^H)\,[n\,(1000 - d\Delta)\{1000 + (F_2 - \delta) D^H\} + 1000 F_2 \Delta]}$$

In the vertical meridian the following equation must be satisfied, as will be again seen by Fig. 4:

$F_C^V - h_2^V - (H_2^V - H_2^H) = F^H - a$, because we suppose that our cylinder corrects the ametropia. Substituting, again we find

$$\frac{F^V\{f^1 - d + H_1^H - H_1^V\}}{A^V + f^1 - d + H_1^H - H_1^V} - \frac{1000\,\delta\, F_2\,(D^V - D^H)}{[1000 + (F_2 - \delta) D^V]\,[1000 + (F_2 - \delta) D^H]} = \frac{1000\, F_2}{1000 + (F_2 - \delta) D^H} - \frac{\Delta F^H A^H}{1000 + (A^H - d)\Delta} \quad \text{(II)}$$

From this equation f^1 can be found. But instead of developing further this equation (II), let us take two special cases, which are of importance for the questions raised before.

The first will be to find the correcting cylinders when they are placed in contact with the cornea, when therefore $d = H_1^H = \frac{1000\,\delta}{n\,(1000 + (F_2 - \delta) D^H)}$. We must remember that the cylinder for the horizontal meridian is always supposed to have been found by actual trial, as it is impossible to find the axial change of the living eyeball in any other way. Calling then Δ the cylinder that corrects the axial ametropia of the horizontal meridian *if placed in contact with the cornea,* we find f^1 or the focal distance of the vertical cylinder also in contact with the eye by equation (II) in

which d is made $= H_1^H$. Doing that in (II) and substituting for A_1^V F_1^V H_1^V H_1^H the former values, we find, after a simple but somewhat tedious calculation, the simple equation

$[\Delta - n(D^V - D^H)] f^1 = 1000$, or $\frac{1000}{f^1} = \Delta - n(D^V - D^H)$. This is then the cylinder that corrects the vertical meridian, while Δ is that for the horizontal one. The whole correction can therefore be written: Δ *sph.* $\bigcirc - n(D^V - D^H)$ *cyl. ax* ‖ meridian of greatest r. We see that the corneal astigmatism of $(D^V - D^H)$ dioptries is corrected at the cornea by a $n(D^V - D^H)$ dioptry cylinder, or the correcting cylinder is n (= 1.3365) times greater than the real corneal difference in refractive power $(D^V - D^H)$.

Let us now for a moment put our problem in the usual way, as it was taken also by Donders. Let us ask what is the cylinder that must be placed at the cornea in front of the *vertical* meridian if it shall bring the parallel rays of light to the same focus behind the cornea as in the *horizontal* meridian, *leaving the lens and retina out of consideration altogether.* Helmholtz's theorem again gives us, if A_1^H $A_1^V F_1^H$ F_1^V have the meaning formerly assigned to them, the value for the new posterior focal distance of the combined system of cylinder and vertical meridian of cornea $= \frac{F_1^V f^1 f''^1}{A_1^V + f''}$. This of course must be equal to F_1^H, and so we have $F_1^H = \frac{F_1^V f''}{A_1^V + f''}$ which, after substituting the former values for F_1^V and A_1^V gives $\frac{1000}{f''} = -n(D^V - D^H)$. This is the same cylinder that we obtained before for the correction of $(D^V - D^H)$, when we took the whole eye with lens and position of retina into consideration, so that for lenses in contact with the cornea the simple assumption suffices very well. But we shall see soon that, though there is no difference between the two cylinders when they are in contact with the cornea, there is considerable difference when we place them at d *mm* from the cornea. At present we must observe that *Javal has made his calculations of the steps so that each corresponds not to* $D^V - D^H$ *but to* $n(D^V - D^H)$; that is, *Javal does not give the real corneal astigmatism independent of a glass; but he gives us the cylinder, which* IF PLACED IN CONTACT WITH THE CORNEA, *will correct* $D^V - D^H$, *i. e.,*

the difference in the refractive power of the two main meridians of the cornea. This is the cause why he makes his steps each 5 *mm.* For we have seen before that $D^{V} - D^{H} = \frac{336.7\,(O^{1} - O)}{2\,n\;281 \times 3} = \frac{O^{1} - O}{6.7}$. Now $n\,(D^{V} - D^{H}) = \frac{(O^{1} - O)\,n}{6.7} = \frac{O^{1} - O}{5}$. If therefore $n\,(D^{V} - D^{H}) = 1$, that is, if there is required a cylinder of 1 dioptry, we find $1 = \frac{O^{1} - O}{5}$, from which is evident that for each dioptry O^{1} must be 5 *mm* larger than before. Javal then is *perfectly right* when he makes his steps 5 *mm* broad, as then each step corresponds to one dioptry increase in the correcting cylinder, but he *is wrong* when he puts on his arc numbers for the refractive power of the cornea which are n times too great. His cylinder, touching the cornea, indeed is $n\,(D^{V} - D^{H}) = n\,D^{V} - n\,D^{H}$, but the refractive power in each meridian is not $n\,D^{V}$ and $n\,D^{H}$ as he makes it, but only D^{V} and D^{H}.

However, it is impossible to wear our cylinders in contact with the cornea, as Javal's calculation supposes. We usually wear them about 14 *mm* away from the eye, about at its anterior focal point. We must now see what changes that makes in the cylinders. Let us then suppose that we put our lenses at the anterior focal distance of the horizontal meridian, and that again we found by actual trial the cylinder Δ which corrects the axial ametropia of the horizontal meridian *at that distance* from the eye.

The cylinder f^{1} (or in dioptries $\frac{1000}{f^{1}}$) for the vertical meridian can then be found from equation (II) by making d in it $= A^{H}$ which gives

$$\frac{F^{V}\,(f^{1} - A^{H} + H_{1}{}^{H} - H_{1}{}^{V})}{A^{V} + f^{1} - A^{H} + H_{1}{}^{H} - H_{1}{}^{V}} - \frac{1000\,\delta\,F_{2}\,(D^{V} - D^{H})}{[1000 + (F_{2} - \delta)\,D^{V}]\;[1000 + (F_{2} - \delta)\,D^{H}]} = \frac{1000\,F_{2}}{1000 + (F_{2} - \delta)D^{H}} - \frac{\Delta\;1000\,F_{2}{}^{2}}{n\,[1000 + (F_{2}\;\delta) - D^{H}]^{2}}$$

Into this equation we put now the values for $F^{V}\;A^{V}\;A^{H}\;H_{1}{}^{H}$ and $H_{1}{}^{V}$ when after reducing and cancelling we get

$$\frac{1000}{f^{1}} = \frac{\Delta - A + \frac{\Delta\,A\,(F_{2} - \delta}{n\,(1000 + (F_{2} - \delta)\,D^{H}}}{1 - \frac{A\,(F_{2} - \delta)}{n\,[1000 + (F_{2} - \delta)\,D^{H}]} + \frac{\Delta\;A\,(F_{2} - \delta)^{2}}{n^{2}\,[1000 + (F_{2} - \delta)\,D^{H}]^{2}}}$$

Here A means $n\,(D^{V} - D^{H})$ that is Javal's astigmatism; so that it shows us the relation between the needed cylinder

and that given by Javal's instrument. Remembering that the cylinder for the horizontal meridian is Δ we can now write for the whole correction:

$$\Delta sph. \bigcirc -A \left\{ \frac{1 - 2\,\Delta \frac{(F_2 - \delta)}{n\,[1000 + (F_2 - \delta)\,D^H]} + \frac{\Delta^2 (F_2 - \delta)^2}{n^2 [1000 + (F_2 - \delta) D^H]^2}}{1 - \frac{A\,(F_2 - \delta)}{n\,[1000 + F_2 - \delta)\,D^H]} + \frac{\Delta\, A\,(F_2 - \delta)^2}{n^2\,[1000 + (F_2 - \delta)\,D^H]^2}} \right\} \text{cyl. (III)}$$

If we further put $\frac{F_2 - \delta}{n\,[1000 + (F_2 - \delta)\,D_H]} = B$ we can write for the cylinder that corrects the astigmatism

$$\text{Cyl} = -A \left\{ \frac{1 - 2B\Delta + \Delta^2 B^2}{1 - AB + A\Delta B^2} \right\} \text{(IV)}$$

It is clear by this formula that the correcting cylinder is not given any more by A or Javal's cylinder but that it depends:

1. On the amount and sign of A as given by Javal.
2. On Δ, the amount and sign of the axial ametropia.
3. On the focal distance F_2 of the crystalline lens.
4. On the distance δ the crystalline lens is placed behind the cornea.

Of these four factors A and can be Δ accurately found by experiment, but to find in each case in practice F_2 and δ would be impracticable. Javal's instrument, therefore, gives us only approximately the correction even for the corneal astigmatism, *though we have supposed the lens to be perfectly spherical.* The amount of discrepancy between our cylinder and that given by Javal must now be determined numerically. In order to do that, we have to make a certain assumption about F_2 and δ. Let us then suppose that F_2 and δ had the values which are given to them by Helmholtz in the second edition of his *Physiological Optics* that is, $F_2 = 50.6$ *mm* and $\delta = 5.7$ *mm*. We will, further, make $D^H = 32.15$ dioptries, which would correspond to a corneal radius of 7.8 *mm*, the average value found by Donders, and finally accept for n, the refractive index of cornea and aqueous humor, the value 1.3365. Then $B = \frac{(F_2 - \delta}{n[1000 + (F_2 - \delta)\,D^H]}$ becomes 0.01375 and $B^2 = 0.00019$. The correcting cylinder then becomes

$$\text{Cyl.} = -A \left\{ \frac{1 - 0.0275\,\Delta + 0.00019\,\Delta^2}{1 - 0.0137\,A + 0.00019\,\Delta\,A} \right\} \text{(V)}$$

The simplest case now will be that in which there is no axial ametropia. Here $\Delta = O$ and our cylinder (V) becomes

Cyl. $= -A\left\{\frac{1}{1-0.0137\,A}\right\}$. As here the influence of Δ is absent; we find rather close approximation to Javal's numbers as the following two tables will show, where a positive A means an astigmatism with the rule and a negative A one against the rule:

A	Correcting Cylinder.	A	Correcting Cylinder.
+ 1	− 1.014	− 1	+ 0.98
+ 2	− 2.056	− 2	+ 1.94
+ 3	− 3.13	− 3	+ 2.88
+ 4	− 4.25	− 4	+ 3.75
+ 5	− 5.37	− 5	+ 4.67
+ 6	− 6.54	− 6	+ 5.54
+ 7	− 7.75	− 7	+ 6.38
+ 8	− 9.	− 8	+ 7.20

Only in 8 D astigmatism as given by Javal, do we have a difference of a whole dioptry, but it will be observed that this difference of a whole D may be positive or negative according to whether our corneal astigmatism is with or against the rule. It is clear from this, that if there is no axial ametropia, only in values of A as high as 4 need there be made any correction. But it is different if axial deviation, and especially a high one, is present. We will show that by a number of combinations, refraining from giving full tables with all the possible combinations of Δ and A and only select a few of the more striking examples. The numbers under Δ are the spheres experimentally found, those under A the numbers given for the astigmatism by Javal's instrument, and the third column gives the full correction as found by formula (III).

Δ	A	Full Correction.	Δ	A	Full Correction.
+ 1	− 3	+ 1 ○ + 2.80 cyl.	− 5	+ 5	− 5 ○ − 6.16 cyl.
+ 1	− 7	+ 1 ○ + 6.25 cyl.	+ 4	− 7	+ 4 ○ − 6.50 cyl.
+ 2	− 2	+ 2 ○ + 1.80 cyl.	+ 6	+ 2	+ 6 ○ − 1.7 cyl.
+ 2	− 3	+ 2 ○ + 2.75 cyl.	− 6	+ 2	− 6 ○ − 2.5 cyl.
+ 2	− 6	+ 2 ○ + 5.25 cyl.	+ 6	− 2	+ 6 ○ + 1.64 cyl.
− 2	+ 3	− 2 ○ − 3.50 cyl.	+ 6	− 8	+ 6 ○ + 6.11 cyl.
− 2	+ 4	− 2 ○ − 4.50 cyl.	+ 7	+ 3	+ 7 ○ − 2.50 cyl.
− 2	+ 6	− 2 ○ − 6.90 cyl.	− 7	+ 2	− 7 ○ − 2.47 cyl.
+ 3	− 2	+ 3 ○ + 1.75 cyl.	− 7	+ 3	− 7 ○ − 3.75 cyl.
+ 3	− 4	+ 3 ○ + 3.50 cyl.	− 8	+ 5	− 8 ○ − 6.6 cyl.
− 3	+ 4	− 3 ○ − 4.6 cyl.	+ 1	+ 5	+ 1 ○ − 5.25 cyl.
− 3	+ 8	− 3 ○ − 9.75 cyl.	− 10	+ 2	− 10 ○ − 2.7 cyl.
+ 4	− 3	+ 4 ○ + 2.5 cyl.	+ 10	− 2	+ 10 ○ + 2.75 cyl.
+ 5	− 3	+ 5 ○ + 2.5 cyl.	+ 10	− 5	+ 10 ○ + 3.5 cyl.
+ 5	+ 2	+ 5 ○ − 1.75 cyl.	− 10	+ 5	− 10 ○ − 7. cyl.
− 5	+ 2	− 5 ○ − 2.4 cyl.	− 20	+ 2	− 20 ○ − 3.36 cyl.

From this table it is evident that in lower degrees of axial ametropia the difference between Javal's astigmatism and the corrected cylinder is not very great, unless at the same time the amount of corneal astigmatism itself is considerable; usually from .25 to .75 cyl., positive or negative, is all the correction necessary. However, in the higher degrees of myopia or hypermetropia, the deviation may be very considerable, amounting even to 2 D or more. Take for example a case, where keratometry shows an astigmatism of 5 D; then this according to the few cases of our table would be corrected by — 6.16 or — 6.6 or + 3.5 or — 7 D cylinder according to the accompanying Δ. We might think then that this corrected value for the cylinder would explain the discrepancy between the keratometric and the clinical findings.[1] But unfortunately it is not so! On the contrary, one frequently finds that the corrected calculated cylinder for the corneal astigmatism is still farther off from the one actually found with the trial-case. Lately I observed a case of myopia of — 7 D, which by two examinations showed a corneal astigmatism of 2 D with the rule and ought to have been corrected by a cylinder of — 2.5 D. Still this case did refuse any minus or plus cylinder and had with — 7.D sph. V = $\frac{6}{6}$. We shall return to this case later on.[2] At present I want to emphasize by it that even our correction by no means always offers an explanation for the difference, but sometimes may even make it greater. *And still it is only these corrected cylinders, that ought to be compared with those found by the trial case, because they are at the same place in front of the eye.*

How then must we explain this divergence? One partial cause of it, it seems to me, lies in the usual way of making the keratometric observations. The patient is simply told to look into the middle of the tube, while the measurements are being made. But what do we do then? We certainly make the observations in the visual line. However, it is not

[1] Sometimes indeed it does so, as in a case observed a few days ago, where Javal showed 1.5 D. Astig. with rule axis 45°. Here was an axial myopia of — 18.D. which would change Javal's 1.5 into 2.5. D. cyl. ax. 45°. She really chose herself — 18.⊂ — 2.75 cyl. ax. 45°.

[2] This case is only one of many others observed in the office of Dr. W. Thomson of Philadelphia, who kindly allowed me to make use of this case for this paper.

the visual line, but the optical axis of the eye passing through the corneal apex, that ought to coincide with the optical axis of the keratometer. For our object is to measure the two main meridians of the cornea and these go through the apex but not through the visual line,—at least not usually. Mostly the angle α is quite a measurable quantity, amounting often to 7°, 8°, and even 10°, and must be considered. It would lead me too far here to explain how it can be done with Javal's or any other good keratometer; it suffices to call attention to this frequent source of error as one that ought not to be neglected. But even if we do avoid this error, we by this do not by any means always get rid of the difference. In his later observations Donders was very careful to measure his meridians through the corneal apex, and still he found frequently a great difference between *A* and the trial-cylinder. In his celebrated book *On the Anomalies of Accommodation and Refraction of the Eye*, 1864, he gives, on page 493, an elaborate table of fifteen cases in which the refraction for each principal meridian was carefully determined by three different subjective methods, then the cornea was measured *in twelve different meridians through its apex*, and from these data the astigmatism of the whole eye and that of the cornea were determined. In none of these cases did the astigmatism for the whole eye agree with that calculated from the cornea, neither in its amount nor in its direction. I shall only select four cases in which I allowed myself to change the denomination of the lenses by focal inches into that by dioptries, and to add one (2d) column to show what cylinder ought to have been placed at the anterior focal distance of the eye; making the assumption that one meridian of the cornea as well as F_e and δ had been normal in the sense before mentioned.

Calculated Corneal Astigmatism.	Corrected Cylinder.	Whole Correction Found by Trial.	Calculated Astigmatism of Crystalline Lens.
3.6 D ax. 63°.	3.25 D	+ 1. ○ + 2.3 cyl. ax. 40°.	2.6 D ax. 173°.
5.2 D ax. 78°.	4.5 D	+ 2. ○ + 3. cyl. ax. 74°.	2.2 D ax. 174°.
1.4 D ax. 148°.	1.25 D	+ 4. ○ + 1.5 cyl. ax. 88°.	1.5 D ax. 174°.
6. D ax. 105°.	5.75 D	− 1.75 ○ + 3.50 cyl. ax. 104°.	2.4 D ax. 18°.

From this table it will be seen that the cylinder calculated for the position in front of the cornea comes a little nearer to the true cylinder in the third column, but is still wide off from the mark. What other causes must be looked for besides the two mentioned? I believe with Donders, that often an astigmatic action of the lens has to be postulated. This action is usually supposed to be due to an oblique position of the crystalline, which indeed has sometimes been observed in cases of astigmatism after a trauma; but I do not see why in the usual cases we should not have resource also to a real curvature astigmatism. *A priori, a curvature astigmatism of the lens with its* TWO *surfaces is just as possible or rather more so than one of the cornea with only* ONE *curved surface* and that of the cornea having been proved to exist, we are justified in assuming one for the lens if necessary. Of course, the cornea having about 32 D refractive power, while the lens has only 20 D, and is besides nearer to the retina, it is clear that the corneal astigmatism must be of greater influence, but that of the crystalline cannot be neglected. This admitted we can then state our problem thus. If in a given case the cornea produces a certain amount of astigmatism A_c that has been exactly calculated, but if actual trial by glasses proves the astigmatism of the whole eye A_o to be different, what must then be the astigmatism of the lens A_l in order to produce together with A_c the observed A_o? This calculation has been given by Donders. But as it involves a knowledge of the curvature of surfaces it would take too much time and space to develop all the formulæ here. Suffice it to say that only when the meridians of the astigmatism of cornea and whole eye coincide, can we say that $A_c + A_l = A_o$, or what is the same, that the astigmatism of the lens equals the difference between that of the cornea and the whole eye. Generally the direction of the axes plays too important a part to allow of that simple formula. This will be apparent by the 4th column of the last table, which was calculated by Donders. The third example is perhaps the most instructive of all, showing that the values of the corneal astigmatism and the whole eye may be almost identical, but still a high lenticular

astigmatism be present, namely, if the axes are very different. It must, however, be added that the lenticular astigmatism, as given in the 4th column, is in fact even higher than as there given, because it is calculated for a crystalline in actual contact with the cornea. But the lens lies about 6 *mm*[1] deeper, and therefore must have a higher astigmatism, in order to produce there the same effect as at the cornea. We see, besides, from the last table, that on the whole the lenticular astigmatism is opposite in its effects to that of the whole eye, so that the astigmatism of the whole eye is almost always much less than it would be if the cornea alone was at fault. This proves, I believe, conclusively, that there is a static lenticular astigmatism in many eyes; for Donders paralyzed the accommodation in these cases, and so did away with any *dynamic* astigmatism. It would be out of place here to discuss this *dynamic* astigmatism, due to an unequal contraction of the ciliary muscle; but I assert that it must be excluded if a mydriatic has been thoroughly used. However, in spite of this evidence for a lenticular astigmatism, it has been asserted by a high authority, that it would suffice to correct the corneal astigmatism as found by Javal's ophthalmometer, without the use of a mydriatic. The question about the mydriatic we will leave undiscussed, though I do not see how then the dynamic astigmatism of the lens, advocated in the same quarter, can be gotten rid of, unless it is supposed that this dynamic astigmatism has its only cause in the corneal astigmatism, and is given up when this has been corrected. However that may be, one thing is certain, that we are obliged to admit a static lenticular astigmatism which cannot be neglected.[2] It must be taken into account by supplementing keratometry by the subjective method, which latter cannot be replaced or superseded by any objective examination. And here it must be stated that even those who assert to correct astigmatism by keratometry alone do not rely upon it altogether; on the contrary, they almost always reduce the kera-

[1] This refers to the distance between cornea and first principal point of the crystalline.

[2] Even Javal himself admits having seen a case of *static lenticular astigmatism of two dioptries.* (*Mémoires d'Ophtalmométrie*, par Javal, p. 121.)

tometric finding by at least 0.50 D. Let us take, *e.g.*, a paper by Dr. Roosa in the *Medical Record* of March 26, 1892, in which he claims to cure many cases of asthenopia by simply correcting the corneal astigmatism. Let us then suppose that every cure was *propter hoc* and not *post hoc*. What does his paper prove? *It simply proves that he does not correct the corneal astigmatism alone.* Take, *e. g.*, Case 8., in which the patient was under the influence of atropine when tested, and in which, therefore, there can be had no resource to a dynamic lenticular astigmatism. Here he finds by the ophthalmometer in O. D. one dioptry of corneal astigmatism with the rule, axis 30° from 90°, therefore at 60°. He gives, however, a + .50 cyl. ax. 45°, therefore .50 D less and with the axis 15° off. In this case we can neglect the correction for the position of the cylinder 14 *mm* from the cornea, as it does not reduce the cylinder to less than 0.95 D. We have therefore to look for another cause, and leaving out of consideration the defects of Javal's instrument, calculation shows that there must be in this case a lenticular astigmatism of 0.62 D with axis at 174° 54′. But as the crystalline lens in this calculation is supposed to be in contact with the corneal apex, it must have a still higher astigmatism in its real position. *We see, therefore, that, far from correcting only the corneal astigmatism, there was also corrected at the same time an astigmatism of the crystalline, amounting to more than* 0.62 *D.* Why then not admit this? Knapp's words, written more than thirty years ago (*Gräfe's Arch.*, Bd. viii., Abth. 2, p. 240), are as correct to-day as they were then. "After one has found the amount and direction of the asymmetry of the eye, the ophthalmometer decides whether the seat of it lies principally in the cornea or in the crystalline lens. For purely practical purposes this is superfluous, for upon therapeutics it has no determining influence."

Keratometry is a valuable method, no doubt, but it is no substitute for the subjective one. This is also well shown in the last July number of the ARCHIVES OF OPHTHALMOLOGY, where Dr. G. Melville Black gives some striking examples of the great discrepancy that sometimes exists between the findings by the keratometer and those by the usual testing. He

gives there, *e. g.*, a case of a patient aged sixty, who in the right eye had $-$ 4.50 ⌒ $-$ 1. cyl. ax. 180° = $\frac{8}{10}$, though the ophthalmometer indicated 2 D astigmatism against the rule, which would have required for its correction either a $+$ 2.40 cyl. ax. 180° or a $-$ 2.4 cyl. ax. 90°. Instead of this it was corrected by $-$ 1. cyl. ax. 180°. This can be only explained by an astigmatism of the lens of more than 3 D with the rule. I repeat, therefore, that the crystalline lens cannot be disregarded in the correction of astigmatism. At the same time keratometry is one of the most valuable methods for a more perfect diagnosis of the actual build of the eye.

It can even teach us something more, to which, as far as I know, attention has never been called before, I mean something about the length of the eyeball. Let me explain by an example. Reference was made above to a case in which there were found 7 D myopia with V = $\frac{8}{9}$ and corneal astigmatism of 2 or even 2.5 D. Here no cylinder did improve the vision, and we are justified in assuming that there was an astigmatism of the crystalline of a little more than 2 D with its axis at right angles to that of the cornea. But the 2 D over curvature of the vertical corneal meridian and the 2 D over curvature of the horizontal meridian of the lens must produce a myopia of 2 D, just as two positive cylinders of each $+$ 2 D with their axis at right angles to each other are equivalent to a $+$ 2 D sphere. The case referred to, which had a normal horizontal corneal radius of curvature, had, therefore, probably only 5 D axial myopia, the remaining two being produced in the way indicated. By the same reasoning, but a little more lengthy calculation, it will also be found in the Case 8 of Dr. Roosa mentioned above, that there is produced a myopia of 0.56 D. One only needs refer to the well-known fact that two cylinders in general are equal in their effect to a new cylinder and a sphere; but the complete formulæ by which the exact quantities can be calculated need not be given here, as they can be found in Donders, *loc. cit.*, pp. 499 and 500. By means of careful observations with a good keratometer, therefore, we get a more perfect understanding of the dioptric apparatus of the eye. It may sometimes even affect our prognosis.

But here must be mentioned another difficulty that is due to the very construction of Javal's instrument. The corneal image has a size of about 3 *mm*, and the area of the cornea used for reflecting the images of the two mires into the telescope is about 0.5 *mm* in breadth, and lies about 1.25 *mm* to either side of the point of the cornea towards which the optical axis of the instrument is directed. If, then, the patient looks into the instrument, or, better still, if he looks so at a point of the large disk that the corneal axis coincides with the axis of the telescope, all we do find with Javal's ophthalmometer is not the radius of curvature (r) of the point looked at, but $r \cos i$, if the cornea was perfectly spherical, and if i is the angle of incidence of the rays which are reflected from the cornea into the telescope. This angle i amounts here to about 10°, and can therefore not be neglected as it can be in Helmholtz's ophthalmometer. Even with a perfect sphere, this circumstance would make the values about two per cent. too great. *But in cases of astigmatism we do not have to do with a spherical cornea but rather with a triaxial ellipsoid, where the formulæ used by Javal for his calculations do not apply.* For in a triaxial ellipsoid each point of the surface has two main radii of curvature, and it depends upon which one of the two focal lines furnished by each point is looked at in order to get the one or the other of these two radii. For example, taking Knapp's first case, in which he determined the ellipticity of the cornea, we find that for a point in the horizontal meridian 1.25 *mm* off from the axis the two principal radii of curvature would be resp. 7.84 and 7.79 *mm*, which if we applied Javal's formula $D = \frac{1000(n-1)}{r}$, would give 43.0 and 43.3, a difference therefore of $\frac{1}{8}$ dioptry. What at the best and by the most complex formulæ we measure with Javal's instrument are the radii of curvature of the cornea at points 1.25 *mm* off from the corneal axis. *But this ring-shaped zone of about* 0.5 *mm breadth is not the whole cornea used in direct vision, a great part of it is left unexplored with the instrument, and it depends therefore upon this remaining area whether the astigmatism observed is little or much deviating from that obtained by subjective methods.*

This difference in the refractive power of the different zones of the cornea has also been justly advocated by Tscherning in Javal's *Mémoires d'Ophtalmométrie* to explain the discrepancy between the diagnosis with and without a mydriatic. It certainly shows that there is no need of the theory of a dynamic astigmatism; but space forbids to enter further into the discussion of this interesting question.

So far we have treated of an eye that contained a lens. Let us now take the rarer but more interesting case of aphakia, where we have only one refractive system.

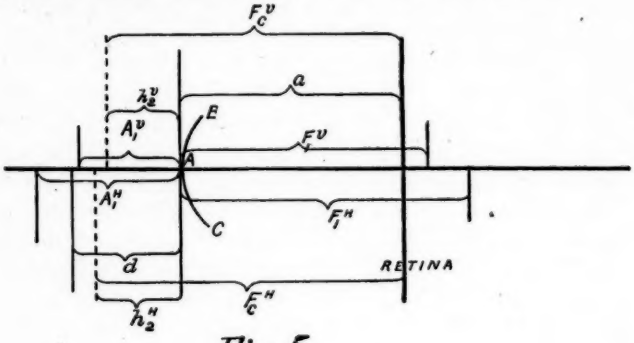


Fig. 5

In the diagram BAC is the cornea, and a the distance of the retina from the corneal apex, while the other letters have the same meaning as before. Suppose we found that a cylinder Δ at the distance d from the cornea did correct the horizontal meridian, then, in the same way as before, by Helmholtz's theorem we easily find

$$\frac{1000}{f^1} = \frac{1000\,\Delta - (1000 - d\,\Delta)\,A\,^1}{1000 - (1000 - d\,\Delta)\frac{d\,A}{1000}}$$

if f^1 is the focal distance of the cylinder that corrects the vertical meridian, and if we put $n\,(D^V - D^H) = A$ as we did before. Writing now our two crossed cylinders as a spherocylindrical combination, we have for the full correction

$$\Delta \text{ sph.} \bigcirc - A \left\{ \frac{\left(1 - \frac{d\,\Delta}{1000}\right)^2}{1 - \frac{d\,A}{1000} + \Delta\, A \left(\frac{d}{1000}\right)^2} \right\} \text{cyl.}$$

[1] This becomes the same formula as that given on page 53, if we put here $d = 0$, as it ought to be.

If we now put $d = 13.75$ *mm*, we get for the correct cylinder not A, as Javal gives it, but

$$-A\left\{\frac{(1-0.01375\ \Delta)^2}{1-0.0137\ A+0.00019\ \Delta A}\right\}$$ which is the same as (V), where a lens was present in the eye. But there is this great difference between the two formulæ, that the first one (V) is only right for certain average values of F_2 and δ, while this last is independent of F_2 and δ and strictly correct if d is made $= 13.75$ *mm*, *i. e.* if the glasses are placed at that distance from the cornea. Cases of aphakia are therefore the ideal field for keratometry, but owing to the high spheres usually necessary, there is a great difference between the correcting cylinder at 14 *mm* from the eye, and that in contact with the cornea, which latter, as we know, is given by Javal's instrument. The following table will show this at a glance, which is given for + 10 D for far, and + 14 D for reading:

A	Δ	Full Correction.	A	Δ	Full Correction.
+ 1	+ 10	+ 10 ○ — .75 cyl.	+ 1	+ 14	— 14 ○ — .66 cyl.
— 1	+ 10	+ 10 ○○ + .73 cyl.	— 1	+ 14	+ 14 ○○ + .65 cyl.
— 2	+ 10	+ 10 ○○ + 1.45 cyl.	— 2	+ 14	+ 14 ○○ + 1.25 cyl.
+ 2	+ 10	+ 10 ○○ — 1.5 cyl.	+ 2	+ 14	+ 14 ○○ — 1.3 cyl.
— 3	+ 10	+ 10 ○○ + 2.1 cyl.	— 3	+ 14	+ 14 ○○ + 1.8 cyl.
— 4	+ 10	+ 10 ○○ + 2.8 cyl.	— 4	+ 14	+ 14 ○○ + 2.5 cyl.
— 5	+ 10	+ 10 ○○ + 3.5 cyl.	— 5	+ 14	+ 14 ○○ + 3.18 cyl.
— 6	+ 10	+ 10 ○○ + 4.1 cyl.	— 6	+ 14	+ 14 ○○ + 3.66 cyl.

This shows conclusively the necessity of correcting in aphakia the keratometrically determined cylinder for its position and for the co-existing axial condition, for even with 1 D it amounts to .25 D and more, while for an astigmatism of 6 D against the rule it amounts to almost 2.5 D. At the same time we observe that in the same individual the cylinder may have to be reduced perceptibly for reading. We also find that the numbers obtained by the keratometer are *always* too high, no matter whether there is astigmatism with or against the rule.

In the following I give a short *résumé* of the main conclusions of this article:

1. Keratometry is a very valuable method for the finer diagnosis of the actual construction of the eye.
2. The cornea having been measured not in the visual line, but in the optical axis of the eye, we find:

a) the direction of the two main meridians of the cornea;
b) the radii of curvature in these two main meridians at 1.25 *mm*, or about 10° to either side of the corneal point looked at, the result being the more incorrect the more astigmatic the cornea;
c) the cylinder that would correct the corneal astigmatism if placed directly in contact with the cornea, and if the remaining part of the cornea not examined by the instrument does not deviate much from the employed area of the cornea;
d) the cylinder that would correct if placed at the usual distance from the eye, as found by formula (V).

3. In cases where the corneal astigmatism differs much from that of the whole eye, especially where there is no A_c, but considerable A_o, we may then find:
a) the axis of the lenticular astigmatism;
b) the amount of the crystalline astigmatism;
c) the sphere resulting from the combined action of the corneal and lenticular astigmatism.

CONGENITAL PTOSIS WITH SYNCHRONOUS MOVEMENTS OF THE AFFECTED LID AND LOWER JAW.

By ALVIN A. HUBBELL, M.D.,

PROFESSOR OF OPHTHALMOLOGY IN THE MEDICAL DEPARTMENT OF NIAGARA UNIVERSITY, BUFFALO, N. Y.

(*With three illustrations.*)

T. W. B. is a boy now seven years of age. He is one of a family of three children whose parents are healthy and intelligent Americans. No malformation is present in any of them, except himself, and none is known to have existed in either the paternal or maternal ancestors. This boy is well developed, healthy, and unusually intelligent. He has a symmetrical and well-shaped head and face, and were it not for the ptosis on the right side he would be quite a handsome lad. Soon after birth it was noticed that the right eyelids did not open with the left, but would always remain closed, excepting when the mouth was opened, and then they would separate and close again with the closure of the mouth. This condition has continued without any apparent change to the present time. On examination I find that when the boy's mouth is closed the right eyelids are also closed, and there is not the slightest movement of the upper lid when the left eyelids are ordinarily opened, or the left eyeball is directed upwards, or when any other movement is made by which the eye or head is turned, or the face drawn, or the lower jaw carried to one side. Figure 1 shows the appearance of the boy's eyes when he is looking forward, with his mouth closed. If the boy is asked to open both eyes as widely as he can, the right upper lid is raised a little, as shown in Figure 2. But this is done entirely by the action of the occipito-frontalis muscle and not in the least by the

levator palpebræ. When, however, the mouth is opened, the right upper lid is involuntarily and unconsciously raised at once. The greater the depression of the lower jaw the higher is the upper lid lifted. In other words, the contraction of the right levator muscle takes place involuntarily with the contraction of the depressor (digastricus) of the lower jaw, and exactly proportion-

FIGURE 1.

ately to that of the latter. This action, however, can be overcome by the orbicularis palpebrarum, whose function is normal on both sides, since the right eye can be voluntarily kept closed while the mouth is opened. When the right eyelids are separated, the eye is seen to be of the same size, and the iris of the same color as the left ; but there is marked divergent strabismus together with

some downward deviation. The eye cannot be turned upwards beyond the median plane, or inwards but little beyond the vertical plane; but downwards and outwards its movements are of usual, or perhaps more than usual extent. The pupil and iris are normal in appearance, size, and reflexes, and are like the left. The refraction of both eyes is, by the ophthalmoscope, emmetropic.

FIGURE 2.

The fundus of the right eye is, like the left, normal in appearance in every respect. But the vision of this eye is amblyopic, being able barely to count fingers at four feet, while that of the left is normal, reading number 6, Snellen, at six metres ($\frac{6}{6}$). Figure 3 shows the appearance of both eyes when the mouth is widely opened.

Remarks.—So far as I know, fifteen cases of this anomaly have been reported. While they have differed somewhat in some particulars, they have all had ptosis on one side with rising of the drooping lid synchronously with some movement of the lower jaw, generally depression. In no case has a post-mortem and histological examination of the brain

FIGURE 3.

been made. Hence the anatomical basis of these anomalous movements is at present merely conjectural. But taking into consideration the fact that the innervation for the levator palpebræ and the digastricus or other muscle of the lower jaw is in these cases undoubtedly one and the same, as shown by the associated movements of these muscles,

and that a part of the nucleus of the descending motor root of the fifth and the nucleus of the root of the third nerve are in close proximity to each other in the neighborhood of the aqueduct of Sylvius, the supposition becomes very probable that an abnormal connection is here established. This abnormal nuclear transposition may vary in different cases, but it appears always to be from some part of the third to some part of the descending root of the fifth nerve. A lack of proper development of the nucleus of origin of more or less fibres of the third nerve appears also to be a prominent characteristic in all cases. In my own case I believe that the origin of the fibres supplying the levator muscle has been transferred from the nucleus of the third nerve to that part of the nucleus of the fifth which sends fibres to the anterior belly of the digastric muscle. The nucleus of the third nerve seems to be undeveloped in that portion from which arise the fibres supplying the levator of the upper lid, the superior rectus, and the internal rectus muscles, while that part sending fibres to the inferior rectus (possibly the inferior oblique) and the iris is normally present. Thus do we get in this case the ptosis, the divergent strabismus, the downward deviation, and the associated movements of the upper lid with the lower jaw.

A CASE OF TOTAL IRIS ABSENCE.

By L. D. BROSE, M.D., Ph.D.,

OCULIST AND AURIST TO ST. MARY'S HOSPITAL, EVANSVILLE, IND.

The mother, Mrs. F., brought her second child, a nine-months-old baby to me, saying ever since its birth she has noticed that it had peculiar eyes. The infant was well nourished and well formed, and save for a side-to-side movement of its eyes, lateral nystagmus, presented nothing out of the way to a non-medical observer. Upon closer examination and with focal illumination a total absence of the iris was found. The suspensory ligament can be seen surrounding the entire lens circumference, while the lens itself occupies a normal position, is of the usual size, and without opacities. With the ophthalmoscope and a plane mirror the fundus is easily illuminated, and a difference in the lens refraction and that through the suspensory ligament distinguished. The eye is hypermetropic, but the degree, as well as more than a fleeting view of the optic nerve and macula region, was denied us, because of the increased eye movements when brought in contact with a bright light, and the tendency of the infant to close the eyelids and bury the organ from the illumination. Neither malformation nor lesion of any kind was seen in any part of the fundus oculi. Tension of eyeball normal. Transverse diameter of the cornea, 9 *mm.* Distance from the inner to the outer canthus, 23 *mm.* The whites of the eyes clear, and the eyes withal, so far as size and development other than above noted, in keeping with what one usually finds in a well developed and well nourished infant at nine months.

Although many of the reported cases of irideremia presented the appearance of total iris absence, yet in the few

cases that have come to a post-mortem examination at least a rudimentary development of the iris was always found. In one case reported by H. Lembeck (*Hirschberg's Centralblatt für Augenheilkunde*, supplement, 1891) the membrane of Descemet had divided into two layers and enclosed the rudimentary iris, which had formed an intimate union with the cornea and sclera beneath the canal of Schlemm. In two additional cases, reported by E. Treacher Collins (*Ophthalmic Review*, March, 1891), the ciliary body formed the rudiment, although so small in size as to have remained undiscovered through the cornea. The proper explanation of this malformation is in all probability that of an intrauterine inflammation involving the iris or its vicinity during the early stages of its development. In support of this view are the pathological changes noted by G. Rindfleisch, assistant in the eye clinic at Heidelberg, and reported in *Graefe's Archiv für Ophthalmologie*. In his specimens, taken from an old lady of fifty-one years, there was a ring-like furrow at the inner side of the corneo-scleral junction of exactly the size of the rudimentary iris just back of it. The possibility of this contact between the iris and furrow-like impression was all the more probable because of traces of previous perforation of the eyeball permitting the aqueous humor to escape, and a partial dislocation of the lens forward, carrying the iris against the cornea, and through pressure retarding its growth. At a later period, when the perforation closed, the eye was fully developed, the re-formation of aqueous restored the lens to its proper place, liberated the iris again, but with no tendency to perfect its development. This inflammatory theory likewise explains why glaucoma is so frequently met with in irideremic eyes, the natural filtration being in part or wholly prevented by the partial or complete closure of the communications between the canal of Schlemm and the anterior eye chamber.

A CASE OF SYMPATHETIC NEURO-RETINITIS WITH PLASTIC IRITIS; RECOVERY; MICROSCOPIC EXAMINATION OF THE ENUCLEATED EYE.

By Dr. T. Y. SUTPHEN, Newark, N. J.

Armin B., a bright, healthy-looking boy, nearly five years of age, was brought to me October 20, 1892. Two months previously, while in the country, he had injured the *right* eye by the thrust of a table-fork. There was only slight inflammation following, and the parents, as well as the physician in attendance, thought the injury was not serious. Within a few days previous to his coming to my office, it had been discovered that the child did not see with that eye. I found the right eye sightless, smaller than its fellow; tension greatly reduced; iris discolored and bulging, and a yellow reflex from the vitreous chamber. In the ciliary region, at the lower border of the cornea, was a sunken cicatrix, while a small gray scar was seen in the cornea, near the temporal side. The pericorneal injection was slight. In the *left* eye there was some photophobia. The ophthalmoscope showed clear media, but a well-defined neuro-retinitis—the neuritis predominating. Immediate enucleation of the injured eye was advised, but the advice seemed so shocking and sudden that the parents declined to accept it without further counsel. Accordingly the child was taken to Dr. Knapp the same day, who, besides advising an immediate operation, urged that a careful microscopic examination of the affected globe and optic nerve should be made, with the view of establishing, in a measure, the theory of the migration of germs as the causation of the sympathetic trouble. The injured eye was removed on the following day Dr. W. S. Disbrow assisting me. Microscopic slides were

made from fluid pressed out from the nerve stump, and the globe immediately placed in a sterilized bottle, and taken to Dr. Knapp for examination. On dilating the pupil of the left eye, strong and broad adhesions were found, which yielded only after several days' use of a mydriatic. The child was kept quietly in bed, in a darkened room, and mercury with chalk administered, in 2-gr. doses, night and morning, for three weeks. The sympathetic inflammation steadily subsided, and when the boy was seen last, on Dec. 30, 1892, all signs of the neuritis had disappeared, and vision was $\frac{20}{20}$. Dr. Ward A. Holden, who kindly made the microscopic examination, and to whom I am greatly indebted, sends the following statement :

"At the inferior sclero-corneal margin is an indrawn cicatrix involving the ciliary body. There has been a general plastic cyclitis, more marked near the cicatrix, with the formation of a fibrous membrane stretching across the ball behind the lens, which has produced a total detachment of the retina and a partial detachment of the ciliary body.

"The choroid in its posterior half, and particularly near the papilla, shows a diffuse cellular infiltration, with small scattered foci of denser infiltration (uveitis maligna, Brailey).

"The intraocular portion of the optic nerve shows beginning atrophic changes, similar to those in the detached retina. Otherwise the nerve and its sheaths are unchanged.

"The examination of cover-glass preparations of fluid from the optic nerve for bacteria was negative. Gelatine tubes inoculated with scrapings of the nerve, after sterilization of the surface, and with fluid from the interior of the eye, were sterile.

"No micro-organisms were found in sections of the eye after hardening in alcohol."

The case is such a clear one, and the examinations for bacteria were so carefully made, that it serves to establish the fact that sympathetic inflammation of the nerve and iris may occur without the existence of migratory germs traceable with the means at our disposal thus far.

ON EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.

By Dr. ANTON ELSCHNIG,

ASSISTANT TO THE UNIVERSITY EYE CLINIC IN GRAZ.

(*With three figures in the text, and two plates, Tab. II. and III. of vol. xxiv., German Edition.*)

Translated and Abridged by Dr. Ward A. Holden.

THE cases to be reported here came under my observation in the Graz eye clinic while this institution was under the direction of my esteemed teacher, Prof. Schnabel.

Case 1.—G. J., a woman of fifty-six, was received Nov. 14, 1887, for a sudden blindness of the left eye, coming on five days previously. She had a moderate-sized goitre, an atheromatous aorta, and hypertrophy of the left half of the heart. The disc and the surrounding retina were white, the retinal vessels very small, the veins on and near the disc pulsating. In the macular region was a circumscribed dark-red patch. V = perception of light.

Nov. 15*th*, at 8 A.M., the retinal opacity is grayish. 10 A.M.—

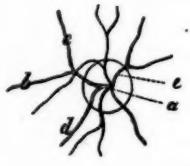


Fig. 1.

A pulsating centripetal movement of the blood column in the principal veins.

Nov. 17*th*, 8 A.M.—At the origin of the inferior nasal artery the red blood column is interrupted by a white patch of cylin-

FIG. 2.

drical form, having a process toward the central artery (Fig. 2, *e*).

Spontaneous venous pulsation; slight arterial pulsation on pressure. The retinal opacity less marked.

Nov. 19*th*.—The median twig of the superior nasal artery shows a white patch near its origin. No arterial pulsation on pressure. At the inferior margin of the pupil a small hemorrhage.

Nov. 21*st*, 5 P.M.—From the central artery short red blood cylinders push into the white superior nasal artery (Fig. 1, *a*) and on into its branches (*b*, *c*). In the course of an hour the movement ceased. Then from the twig *b* the blood passed centripetally, partly into the main branch, partly into the twig *c*; later the blood in the large artery *a* moved centrifugally into the twig. This process was frequently repeated. Pulsation in veins. 7.30 P.M.—Pulsation in central artery; *a*, bloodless; *d*, filled with blood. Veins small; no pulsation.

Nov. 22*d*, 8 A.M.—Marked pulsation in the central artery; the superior nasal artery empty. 10 A.M.—*a*, filled with blood, which moves rhythmically in the normal direction. 11 A.M.—In *a* no movement of the blood and none in the central artery. The entire circulation stops suddenly for a time and then begins again. In the periphery the pigment epithelium shows light patches.

Nov. 23*d*.—Circulation more active, but only in the arteries. The arteries are all small near their origins, and are normal in size or larger farther out. Arterial pulsation on pressure. The disc dirty-gray, the inner and lower margins sharp. The retina clearer in its nasal portion, temporally and particularly about the macula still opaque.

Dec. 7*th*.—On the disc, which has an opaque yellowish-white color, a loose gray tissue has formed in the supero-temporal quadrant, and projects into the vitreous, carrying fine vessels and vascular networks. This tissue increases in the following

days. The red spot in the macula loses its sharp contour, and becomes stippled with white.

Dec. 19*th*, 20*th*, and 21*st*.—Circulation of an interrupted or granular blood column clearly seen in the twigs of the upper papillary artery.

Dec. 22*d*.—With diastole a blood column pushes from the central artery into the upper papillary artery *e*, and returns in a granular condition.

The nasal half of the disc assumes a more bluish-green color and a transparent appearance ; the vascular formations increase. The white patch in the inferior nasal artery remains unchanged. Some arteries become thread-like, so that it is impossible to follow them. In the neighborhood of the disc to the temporal side there are striated hemorrhages. The dull-yellow patches in the pigment epithelium have appeared near the disc.

When discharged, four months after her admission, the disc had a sharp outline, was white, and in the centre greenish, the vessels narrow, an embolus in the inferior artery, a fine network of vessels covering the entire disc ; the wall of the arteries on the disc all thickened ; the pigment epithelium of the nasal half of the retina irregular, and showing numerous round yellow spots reaching to the disc ; the macular region more darkly pigmented, but showing the same changes as the remainder of the fundus ; the remains of numerous hemorrhages near the disc ; pulsation of the papillary vessels on pressure.

Case 2.—*June* 17, 1890, I examined in the medical department, T. H., a woman of fifty, who was somnolent, aphasic, and hemiplegic. The pupils are of equal size, the left irresponsive to light, but reacting consensually. The nasal and inferior margins of the disc are indistinct, the retina pale but transparent, and almost lead-colored near the macula. The superior papillary artery is well filled, the other retinal vessels are small. The inferior artery is simply a white cord, in the twigs of which a small blood column reappears. The veins are thin everywhere. The right eye normal. The patient had a hypertrophied heart of irregular action, and albuminuria. On June 13th she became suddenly unconscious, right hemiplegia with facial paralysis, hemianæsthesia (slight), and motor aphasia. This condition has not changed. The patient before this attack had not complained of her sight.

June 18*th*, 10 A.M.—The nasal portion of the disc is covered

with a thick white cloud. The inferior papillary artery is quite white, its twigs only here and there containing blood. The superior papillary artery and its branches are filled with blood, and each branch of the first order is larger in calibre than the main artery. The blood column is continuous. The veins as dark-red cords without a reflex may be followed uninterruptedly to their junction in the central vein. No small papillary vessels visible.

In the macula there is an oval cherry-red, fairly sharply contoured spot.

June 18th.—The cloudiness of the disc and retina is increased. An interruption of the blood current appeared at times in the superior artery and its branches. Some of the larger arteries show a thickening and cloudiness of their walls.

June 19th.—The superior and inferior papillary arteries are still white. The superior retinal arteries are well filled and tortuous, the inferior narrow, and the blood column interrupted. Very strong and repeated pressure on the ball produces scarcely a change in the blood cylinders, and there is no sign of pulsation. Formation of cholesterine in the macular region. In the veins the column is granular, and moves in a centripetal direction with a varying rapidity.

June 20th.—Disc whiter and more cloudy. The papillary arteries have a very thin blood column, no circulation in the veins.

Cloudiness of retina less marked, and calibre of the vessels increased in the days following. The macular spot becomes darker and irregular in form, and loses its sharp outline.

July 8th.—The narrowing of the inferior papillary artery extends to its temporal branch which ½ disc diameter from its origin regains its normal calibre. The light patches in the macula change from day to day, and resemble more and more the changes found in albuminuric retinitis, without having so regular an arrangement. Arterial and venous pulsation can be produced by pressure.

Aug. 3d.—The disc white, outline sharp. The retinal veins of normal size. The arteries almost obliterated on the disc, but of nearly normal calibre in the periphery. The light spots in the macular region cannot be clearly seen, and are scattered over the retina, giving it a variegated appearance. On this day the patient died.

Autopsy.—Between the thalamus and corpus striatum and the internal capsule, the cerebral substance shows a necrotic softening. The same change is found in the posterior portion of the lenticular nucleus and in a small portion of the external capsule. Internally the focus reaches the wall of the third ventricle. The vessels of the base of the brain are very rigid. The walls of the vertebral artery are so thick as to almost obliterate its lumen. The arteria fossæ Sylvii is completely blocked at its origin with a red thrombus, beyond which it is empty. The heart is greatly enlarged, the muscles lax; the aorta thickened. Diagnosis: encephalomalacia from thrombosis of the arteria fossæ Sylvii, atheroma of the cerebral arteries, dilatation of the heart.

The contents of the left orbit, with the entire optic nerve, and the internal carotid artery from its exit from the carotid canal, and the right eye and optic nerve, were removed and hardened in Müller's fluid.

The macular region was removed from the rest of the ball, and then the nerve, retina, and choroid were cut into small pieces, some of which served for teased preparations, the others being cut in serial sections in various directions. Preparations were stained with hematoxylin-eosin, picrocarmine, and according to Pal's modification of Weigert's method.

Microscopic Examination—Vessels.—The lumen of the internal carotid at its emergence from the carotid canal is filled with a thrombus which rests on the vessel walls, and which has a coarsely granular appearance, being composed partly of concentric layers and partly of anastamosing threads of homogeneous material and irregular globules. At points the thrombus does not rest on the wall, and the free lumen so left is filled with closely packed blood corpuscles. The vessel wall shows an atheromatous degeneration.

The central end of the thrombus could not be followed in the carotid canal, but peripherically it extended nearly to the origin of the ophthalmic artery, and from this point on into the ophthalmic artery a thrombotic mass adherent to

the vessel wall narrowed the lumen without obliterating it. This latter portion of the thrombus is of later date, and the corpuscles composing it are better preserved.

The ophthalmic artery and its direct branch, the central artery of the retina, have a free lumen, the intima being thickened in places by the presence of a striated tissue with spindle-formed nuclei, a change often found—particularly in the optic canal—in old persons.

The central artery enters the nerve 10 *mm* from the sclera, and passes to the centre, where it runs directly to the ball. Just after its entrance into the nerve it gives off a large vessel, which divides, one branch accompanying the central artery to the lamina cribrosa, and the other bending backward and breaking up into a great number of twigs which run in the connective tissue between the nerve bundles (the artery first described by Vossius, *Arch. f. Ophth.*, vol. xxix., 4, p. 143).

The central vein divides into its principal branches behind the lamina cribrosa; the central artery divides into the superior and inferior papillary artery near the surface of the disc, after having previously given off a number of small papillary twigs. In the intrabulbar portion of the artery there are the same changes that were found in the ophthalmic artery, which were also found in the artery of the normal right eye. Within the ball the central artery has a narrow cleft-shaped lumen, the delicate endothelium is regular, the elastica folded in the normal manner.

The first portion of the inferior papillary artery resembles the central artery, its middle portion showing the following characteristic changes. The lumen is narrowed by an irregularly spindle-shaped deposit on and in the walls.

Peripheral end of the new formation (Fig. 1, Plate II.). Diameter of artery, exclusive of adventitia, 0.12 *mm*. Muscularis and elastica well preserved. The lumen is contracted to a kidney form by a sharply contoured formation, which is separated from the elastica by a thick layer of delicate fibres, between which lie proliferation cells and slender intima nuclei. The formation rests on the lateral anterior wall of the vessel like a gall on an oak leaf. The surface of

the formation toward the lumen is covered with an endothelial layer which is continuous with the endothelium lining the remainder of the vessel wall. The endothelial layer is partially detached from the thickened intima, but this is a post-mortem change.

The formation is from 0.026–0.035 *mm* in diameter, stains faintly with eosin, and scarcely at all with carmine or hematoxylin. Highly magnified, its periphery is smooth, containing a few nuclei from the intima, its centre coarsely granular.

The central end of the formation (Fig. 2, Plate II.) is more complicated. The lumen of the vessel is eccentric, elliptical, 0.025–0.033 *mm* in diameter, lined with an endothelial layer. The deposit here is larger than in its peripheral portion. In its superficial layers, on all sides, are cells, partly identical with the intima cells, partly similar to young connective-tissue cells. By a proliferation of the intima on either side of the deposit, the sharp angles seen in Fig. 1 are obliterated, and the lumen is not kidney-shaped, but elliptical.

The elastica is well preserved; the regular stratification of the cellular layers of the muscularis is somewhat interrupted by the presence of a finely fibrous tissue; the adventitial connective tissue is sclerotic, with here and there isolated or grouped round cells.

The last portion of the inferior papillary artery shows a free round lumen, a normal muscularis, a thickened intima, and groups of round cells in the sclerotic adventitia.

The superior papillary artery is in many respects similar to the inferior. Besides the changes in the walls, there is a slight deposit on the intima, bounded by endothelial cells.

Twigs of the Inferior Papillary Artery.—Near the margin of the disc the papillary artery divides into a temporal and a nasal twig. The thick connective-tissue sheath accompanying the central vessels becomes thinner after the division, but forms a fairly thick adventitial sheath for the branches. The lumen is of normal size and free, the muscularis and endothelium of normal appearance, the muscularis separated

from the connective tissue of the adventitia by groups of small round cells.

Just beyond the margin of the disc, a marked change appears in the inferior nasal artery. It is bounded from the retinal tissue by a thick layer of round cells, and its walls are sclerotic. In place of the endothelium there is a loose delicately meshed tissue, not smoothly bounded toward the narrowed lumen. The smaller twigs are sclerotic, and some of them are obliterated.

Twigs of the Superior Papillary Artery.—The superior papillary artery divides near the margin of the disc into a small nasal artery and a large temporal artery. The temporal artery is sclerotic. The eccentric narrow lumen is surrounded by a layer of dense connective tissue, with numerous spindle-formed nuclei, and between this and the tunica propria is a layer of loosely meshed connective tissue.

Macular Region.—Since the arteries and veins of this region show more marked changes than in the remainder of the retina, they will be described by themselves.

1. *Upper Macular Region.*—The arteries here are so changed as to be scarcely recognizable. The muscularis is wanting; the outer sheath, sharply limited from the surrounding retina, consists of a finely fibrillar connective tissue, which replaces the muscularis. Internal to this sheath is a layer two or three times thicker, containing a few intima cells and many round cells, lined with endothelium, and enclosing a lumen of varying width. The lumen contains closely grouped but normally staining blood-vessels. The spongy character of the tissue, which has apparently developed from the intima, is rendered clearer by the presence of blood corpuscles in it. There is no sharp separation of the two layers by an elastic membrane.

Between the two arteries running to the macula is a larger oval vein. Its walls consist of loose, wavy connective tissue inserted between the outer and the inner endothelial layer.

2. *Lower Macular Region.*—The inferior nasal artery is best preserved. It has a wide lumen, a normal or slightly thick-

ened endothelium, which is separated from the sclerotic propria by the wrinkled elastica. Nearer the periphery there are numerous cellular plaques between the elastica and the inmost endothelial layer, and round cells in and near the outer sheath. Two millim. from the macular elevation is an artery, 0.06 *mm* in diameter, which is almost entirely obliterated (Fig. 3, Plate II.).

The folded propria consists of concentric layers of sclerotic connective tissue. The contents of the vessel consist of a cellular spongy tissue with blood corpuscles.

Peripherically, the chronic inflammatory changes in the vessel wall are more marked than near the disc. The arteritic plaques become more numerous, and near the macula the smaller vessels are blocked by an endarteritis.

Résumé.—The central artery of the retina and the ophthalmic artery have a free lumen, and show no other pathological changes in their walls than are found in the well eye. In the inferior papillary artery there is a plug which has retained its original character at its peripheral end only, being here, however, covered with endothelium, and at its central end is in an advanced stage of organization by the proliferation of the intima and the migration of cells into the thrombus. This cannot be considered as an atheromatous plaque, but is certainly an embolus. The same may be said of the analogous changes in the superior papillary artery. Here the embolus was originally smaller, and is now more highly organized than in the inferior papillary artery.

The retinal arteries and the peripheric veins show less marked chronic inflammatory changes, which may be called arteritis and phlebitis prolifera.

The collections of blood corpuscles in the new-formed connective tissue are to be explained as having passed through small ruptures in the endothelium into the spongy tissue.

The Papilla.—The temporal half consists of connective-tissue bundles continuous with those of the lamina cribrosa. There is no trace of nerve fibres to be seen. The nasal half projects nearly twice as far above the choroidal plane as the temporal half. The lamina cribrosa is convex backward.

The papillary tissue retains its normal structure in so far as the preservation of the supporting substance of some of the nerve bundles, but has no nerve fibres. Everywhere there are numerous vessels of larger calibre than normal entering the disc with the connective-tissue tracts; and, besides these, there are a number of sclerotic twigs from the central artery.

The disc passes over into the retina in the following manner (Fig. 7). The loose connective tissue, with numerous glia cells forming the inmost portions of the disc, is arranged parallel to the surface of the disc, and continues, as delicate tracts, into the retina, exactly as if the nerve-fibre bundles were present.

Temporally where all the layers of the retina are present, the retina does not end where the choroid ends, but pushes over into the disc. On the nasal side all the layers of the retina end regularly at the margin of the disc.

In a small crescent-shaped portion of the retina near the disc, all the layers can be distinguished. Beyond this portion the retina consists of five layers only (Fig. 3, Plate II.). The internal layer consists of the nerve-fibre, ganglion-cell, and inner reticular layers. The inner nuclear and all the outer layers are preserved.

I.—The inner layer (Fig. 3, 1) consists of Müller's fibres and nuclei; the cavities are filled with granular albuminous masses. No ganglion cells can be found.

II.—The inner nuclear layer (Fig. 3, 2) consists of a single stratum of elliptical pale granular nuclei, belonging to the supporting fibres of Müller.

III.—The outer reticular layer (Fig. 3, 3) is not sharply bounded, and is of varying thickness. In some parts it is quite thin where the nuclei of the outer nuclear layer have pushed through until they have reached the nuclei of Müller's fibres.

IV.—The outer nuclear layer (Fig. 3, 4) has its natural arrangement, but is rarefied, as the nuclei have pushed into the reticular layer.

V.—The rods and cones show only the ordinary post-mortem changes.

The changes in the retina in general may then be said to be the disappearance of all the nervous elements of the cerebral layer of the retina.

The Fovea.—In place of the usual fovea with elevated margins, there is a round patch about the size of the disc, sunken, smooth, of dark-brown color, adherent to the choroid, and not detaching readily like the remainder of the retina.

A fovea centralis does not exist. The entire thinned portion has a thickness of 25–40 μ, and consists of two layers without a sharp line of demarcation. The inner layer is composed of a delicate network of fibres similar to Müller's fibres, but crossing in various directions and mostly running parallel to the surface of the retina, having pale oval nuclei and numerous small very thin-walled vessels.

The outer layer is composed of dark homogeneous nuclei similar to the elements of the outer nuclear layer, and epithelioid cells crowded for the most part closely together. In the centre the limitans externa and the rods and cones are wanting. The cells lie free on the outer surface of the retina, and among them are found pigment cells entire or in fragments.

The region of the fovea consists, then, of a richly vascular connective tissue, fibrous in its inner portion and rich in cells in its outer, in which there are remains of the elements of the outer nuclear layer.

The retina is best preserved in its upper half, where the circulation was soonest re-established. Otherwise the changes are fairly uniform in the entire retina, excepting a portion of the nasal and a portion of the temporal æquatorial region.

After the careful removal of the retina, the choroid is seen to be covered with a somewhat irregular pigment epithelium. In its lateral portions the pigment epithelium is covered with a delicate albuminous membrane (Fig. 5, *a*). The pigmentation of the epithelium is very irregular, and particularly so where the albuminous membrane is thickest (Fig. 5, *p* at left).

There are two degenerated patches in the retina near the equator of the ball, one to the temporal and one to the

nasal side. The margins of the atrophic patches (Fig. 6) consist entirely of the connective-tissue portion of the retina. The rods and cones are wanting. The pigment epithelium, partially separated from the lamina vitrea by the albuminous membrane, consists of round and irregular cells, not uniformly pigmented. In the central portion of the atrophic patches the pigment epithelium does not exist as an epithelial layer. There is here a layer as thick as the remainder of the retina, consisting of spindle-formed cells which have lost their epithelial character but retain some pigment needles. In some portions this proliferated epithelium cannot be separated from the atrophic retina where the limitans externa is wanting. Over this proliferated epithelium in some parts is a distinct vitreous membrane, so that the whole comes to resemble, in a measure, a capsular cataract (Fig. 6, *p*). The preserved portion of the retina passes quite suddenly into the atrophic patches (Fig. 5).

The choroid is normal, except for the ordinary changes of age, a moderate sclerosis of the vessels, and a slight increase of nuclei in the layer of middle-sized vessels.

The optic nerve is atrophic, the nervous tissue replaced by connective tissue. The atrophy of the nerve is less marked a little distance behind the lamina cribrosa, and posterior to the entrance of the vessels it is normal.

PREVIOUS LITERATURE.

The uncomplicated cases of embolism of the central artery, which have been anatomically examined, are as follows:

1. GRAEFE[1]-SCHWEIGGER[2]—Death one and a half years after ophthalmoscopic diagnosis of embolism. The central artery completely blocked just behind the lamina cribrosa. Cadaveric changes too marked to permit further examination.

2. SICHEL[3]—A patient with a mitral insufficiency becoming suddenly blind, and the vision then clearing up, except in a central scotoma, showed a slight dilatation of the veins; arteries of normal calibre; an extravasation at the macula, and white plaques in the retina. Four months later, the vision was rapidly reduced to perception of light. The veins were hyperæmic; the arteries degenerated to white cords for a short distance from the disc, and then regaining their calibre; in the macula, a reddish-brown patch with small white

[1] *Graefe's Archiv.*, vol. v., 1.
[2] *Vorlesungen über den Gebrauch des Augenspiegels*, p. 140.
[3] *Arch. der Physiol.*, vol. iv., p. 83, 207.

spots. The eye was put into Müller's fluid sixty hours after death. Leber believed that there had been a partial blocking of the central artery, and later a complete thrombosis. The eye was not well preserved, and, furthermore, the case was not one of simple embolism.

3. PRIESTLEY SMITH [1] published a case in which the examination was equally incomplete. The central artery was changed into a connective-tissue cord in the optic nerve, four months after the sudden blindness.

4. SCHMIDT-RIMPLER [2]—A man of forty-eight, with complicated vitium cordis, became suddenly blind in left eye after a left hemiparesis. Twenty-four hours later, the typical picture of embolism of central artery. From the following day, chemosis, exophthalmus, irido-choroiditis, with exudation in the anterior chamber and vitreous. Four months later, small irresponsive pupils; posterior synechiæ; atrophy of optic nerves with small vessels; symptoms of chorio-retinitis, and retinal hemorrhages. Autopsy, ten months after blindness. Central artery and accompanying branch, given off at its entrance into nerve, blocked by a yellowish hyaline plug, leaving only a small lumen free. Atrophy of disc, retina, and choroid.

5. F. POPP [3] examined the left eye of a woman of sixty, who had lost her sight three years previously from embolism of the central artery. Optic nerve, nerve-fibre and ganglion-cell layers atrophic. Retina otherwise normal. No obliteration or narrowing of the lumen of the arteries.

6. GOWERS [4]—A man of thirty, with a bicuspid insufficiency. Five days after, a sudden right hemiplegia, with disturbances of speech and of consciousness; ophthalmoscopic examination showed in the left eye the picture of embolism of the central artery. Autopsy, two months later. Here and there in the vessels of the nerve and disc are small granular masses and a large plug in the central artery, not completely blocking it, one-eighth inch behind the lamina cribrosa. The fibres of the nerve showed a beginning degeneration.

The nerve-fibre layer of the retina is somewhat swollen in the neighborhood of the papilla. The remainder of the retina is infiltrated with small nuclei, similar to the elements of the nuclear layers. Macular region: at some points all the retinal layers filled with nuclei, the supporting fibres covered; at other points the fibres of the outer reticular layer separated, as in simple œdema; the nuclear layers normal; again, at other points, the nuclear layers increased in thickness, and in place of the rods and cones are nuclei, the pigment epithelium being in close contact with the outer nuclear layer.

7. HIRSCHBERG [5]—Man of forty-five, with vitium cordis, embolism of right central artery. Complete re-establishment of the circulation. Death eighteen months later. Atrophic excavation; nerve fibres and ganglion cells wanting. Central artery and vein within the nerve entirely normal. The orbital portion of the nerve almost normal.

8. SCHNABEL and SACHS [6]—A man of thirty-three, with a heart lesion, became suddenly blind in the left eye. The following day the typical picture of em-

[1] *Brit. Med. Jour.*, April 4, 1874.
[2] *Archiv für Ophth.*, vol. xx., p. 287.
[3] Inaug. Dissert., Regensburg, 1875.
[4] *Lancet*, Dec. 4, 1875.
[5] *Centrabl. f. prakt. Augenh.*, Jan., 1884.
[6] *Arch. f. Augenh.*, vol. xv., p. 24.

bolism of the central artery was found. Nine hours later, the calibre of the artery was normal, or nearly so; in the inferior papillary artery, a white spot; in most of the veins normal circulation. Three months later, death. In the intra-laminar portion of the central artery, an embolus covered with several layers of endothelium, partially blocking its lumen, and a similar embolus in the inferior papillary artery. Some small retinal arteries show a marked thickening of their walls with a narrowing of the lumen. The optic nerve from chiasm to ball is thinner, and shows a beginning degeneration of the fibres. In the retina the nerve-fibre and ganglion-cell layer is wanting; the inner nuclear layer is thin; the outer nuclear layer and the rods and cones are normal.

The cases of NETTLESHIP (two), LORING, and ROTHMUND-EVERSBUSCH are more complicated.

Remarks.

Ligation of a single carotid artery does not affect vision or produce any marked change in the retinal circulation, since the circulation is at once restored through the medium of the circle of Willis. For this reason, thrombosis of the internal carotid cannot directly affect vision. It is possible, however, that thrombosis of the carotid and ophthalmic arteries is a frequent source of embolism of the

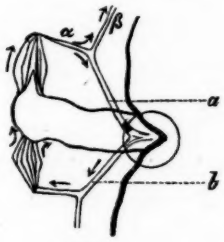


FIG. 3.

central artery, for it is difficult to believe that every embolus in a retinal artery has been carried there from the heart.

The absence of an embolus in the anatomical preparation does not prove that the clinical diagnosis was incorrect.

If some time has elapsed, there may be only a connective-tissue deposit on the intima, which may be covered with endothelium, and escape observation in a casual examination.

Schnabel, from his clinical observations and anatomical examinations, has concluded that the primary anæmia or

ischæmia is due to two causes: the embolus itself, and the contraction of the arterial walls which it causes. When the contraction passes off, the blood again circulates in the channel which is not then completely blocked by the embolus. On this point I am in accord with Schnabel.

The centripetal direction of the arterial blood current may be explained as follows. The arterial trunk *a* contains an embolus. The blood in its twigs and capillaries, when stasis takes place, is under a pressure not exceeding that of the intraocular tension. The neighboring trunk *b* is free, and its circulation is normal. A vein of the inferior system anastomoses at the ora serrata (Leber, Schwalbe) with a vein of the arterial twig α. As the tension in α is less than in the other, the blood flows back into α, and passes down into *a*, and also out centrifugally in the arterial branch β, the tension of which is also low.

The cherry-red color of the macula is partly due to the contrast with the œdematous portion of the retina, but the changes which it undergoes are due to the changes in the pigment epithelium, consisting first in an increase of pigment, and later in its disappearance. These changes in the pigment epithelium are probably not due directly to the retinal anæmia, but to the collateral hyperæmia of the choroid.

Explanation of the Drawings.

FIG. 1. Transverse section of the inf. papillary artery; peripheric end of the embolus. Reichert, obj. 5, ocular III.

FIG. 2. Transverse section of the inf. papillary artery; central end of the embolus. Obj. 5, oc. I.

FIG. 3. Retina between macula and disc; vertical meridional section. Obj. 5, oc. III.

1. Inner layer (nerve-fibre, ganglion-cell, and inner nuclear layer).
2. Nuclei of Müller's supporting fibres (inner nuclear layer).
3. Outer reticular layer.
4. Outer nuclear layer.
5. Layer of rods and cones.

FIG. 4. Vertical meridional section through the centre of the disc. Obj. 3, oc. III.

FIG. 5. Horizontal meridional section through the temporal portion of the retina anterior to the equator. Obj. 3, oc. III. *p*, pigment epithelium; *a*, layer of coagulated albumen; *r*, retina.

FIG. 6. Section through the retina and choroid at the temporal atrophic plaque. Obj. 5, oc. III. *r*, retina; *p*, tissue from proliferation of pigment epithelium; *ch*, choroid.

FIG. 7. Optic papilla, upper half. Obj. 2, oc. I.

ON EMBOLISM OF A BRANCH OF THE CENTRAL ARTERY OF THE RETINA.[1]

By Dr. WARD A. HOLDEN.

(*With five figures in the text.*)

IN the month of December last the rare opportunity was offered me of examining five undoubted cases of embolism of a branch of the central artery of the retina. Four of these occurred in the private practice of Dr. Knapp, to whom I am indebted for the permission to publish them.

The few remarks following a short report of the cases will be confined to a consideration of the relation of the visual field to variations of arterial distribution, and to the manner of restoration of the interrupted circulation.

Case 1.—A man of fifty-one, noticed eight days previously a sudden blindness in his left eye which soon after regained its sight. For two days following, the vision of the eye vacillated and then within ten minutes became almost completely abolished, which condition has remained. V = $\frac{1}{200}$, eccentric, in a small field just to the temporal side of the fixation point, corresponding to an oval portion of the retina between disc and macula. The retinal arteries are somewhat contracted, the veins dilated, the retina œdematous except in its periphery and in a small area between disc and macula, in which area runs an artery whose circulation is apparently uninterrupted. There is no heart murmur, but he has been under treatment for an amyloid kidney due to syphilis. Fig. 1.

Case 2.—A man of twenty-three, eight months ago, while taking violent exercise, noticed a sudden complete blindness in left eye. The following day the lower half of objects could be seen. This

[1] Read before the section in ophthalmology N. Y. Academy of Medicine, Jan. 16, 1893.

condition has remained. V = $\frac{20}{20}$—. The field shows a superior hemianopsia, the blind portion encroaching somewhat on the *inferior* half of the field to the temporal side. The inferior branch of the central artery is small and does not pulsate on pressure. The upper third of the disc is of normal color, the middle third is pale, the lower third is blue and excavated. He has no heart murmur, but there is a history of albuminuria. Fig. 2.

CASE 3.—An unmarried woman of thirty-four a year ago noticed that with the right eye she could not see the upper half of objects. V = $\frac{20}{20}$. Field shows an irregular superior hemianopsia encroaching upon the inferior half to the nasal side, and to the temporal side reaching 15° from the median horizontal line. The inferior retinal arteries are small, some of them being almost entirely obliterated. Each branch, however, near the margin of the disc receives an anastomosing branch arising either from a superior retinal artery or from a ciliary vessel, and beyond this point is fairly well filled. The upper third of the disc is normal in color, the middle third is white and atrophic, the inferior third has a slight reddish tint where branches of cilio-retinal vessels are seen. Patient has a valvular heart lesion. Fig. 3.

CASE 4.—A married woman of thirty-three on awaking seven days ago noticed a diminution of sight in the left eye, which has remained constant. V= $\frac{4}{200}$ eccentric. The field shows a scotoma occupying the infero-nasal quadrant and including the fixation point. An embolism is seen in a horizontal branch of the superior papillary artery, which divides, sending two twigs toward the macula and a third upward. The retina about the disc is somewhat œdematous. The disc itself is normal in color. There is no heart lesion or albuminuria, but the patient has a history of syphilis, complains of severe headaches, and has a small, weak pulse. Fig. 4.

Nine days later: the œdema of the retina has disappeared. The macular region is studded with punctate glistening dots; the temporal half of the disc is atrophic. Field as before.

CASE 5.—A man of thirty on awakening five weeks ago noticed that he could not see the upper portion of objects with the right eye. Condition has remained constant. V= $\frac{20}{20}$. Field shows a hemianopsia superior with sharp inferior limit passing nearly through fixation point. Inferior branch of central artery small. Inferior two-thirds of disc pale. Valvular heart lesion. Fig. 5.

The Branches of the Central Artery.—The central artery of the retina divides just before or just after its emergence in the disc into a superior and an inferior branch. Each of these two branches in its turn divides near the margin of the disc into a temporal and a nasal branch. So we might suppose that embolism of a branch of the first order would cause a sharp superior or inferior hemianopsia, and embolism of a branch of the second order a quadrant scotoma. The matter, however, is not so simple. In many eyes there is also a small vessel, the median artery, running horizontally to the nasal side, and often also one or two small arteries running horizontally to the temporal side, toward the macula. These small horizontal vessels may, some or all, be given off the main artery low down before its bifurcation. Or they may arise from the branches of the first or second order, in which case the nasal artery usually arises from an inferior branch, and the temporal from a superior.

Taking a case in which a horizontal artery is given off below the bifurcation, if there be an embolism between this branch and the bifurcation, there will be blindness, except in a small portion of the retina supplied by this branch. Such an instance is Case 1, in which only a small macular branch is free.

Taking a case in which a lateral artery is given off from a superior or an inferior branch, if there be an embolism of this branch, the hemianopsic blind portion of the field will extend over the horizontal median line on the nasal or the temporal side, as the case may be. Such an instance is Case 2, in which a median nasal artery arises from the inferior branch, which is blocked, and to the nasal side a portion of the superior half of the retina is blind.

Taking a case in which a lateral artery is given off from a superior or an inferior branch, if there be an embolism of this artery only, there will be a scotoma varying with the distribution of this artery. In Case 4 the temporal horizontal artery which is blocked divides into two branches running to the macula, and one running upward, and there is a quadrant scotoma including the macula. The fact that the scotoma extends to the periphery makes it appear doubtful

whether the superior temporal artery, which was free when the case was first seen at the seventh day, may not have been previously blocked for a time.

So far the relation of the field to the vessels blocked is fairly clear. There are, however, peculiarities in regard to the periphery of the field which are not so readily explained. It is a common experience to find in cases of embolism of the main artery a bit of peripheric field preserved, yet such small arteries as may be given off before the bifurcation of the main artery are invariably short, and do not extend to the periphery. These cases have been explained by some as being due to an incomplete blocking of the artery. Others have thought that the periphery of the retina was supplied by anastomosis with choroidal vessels at the ora serrata. In Case 2 we find a peripheric area beyond the blind portion, in which vision is preserved, apparently indicating that the temporal periphery of the inferior half of the retina is supplied by the superior vessels. The reverse of this condition is shown in Case 3, in which, far to the nasal side of the field, the blind portion passes beyond the median horizontal line, apparently indicating that the blocked inferior vessels have supplied a portion of the superior half of the retina to its temporal side.

The Re-establishment of the Circulation.—According to Leber's description, the optic nerve far back is supplied with vessels from the pial sheath. After the entrance of the central artery into the nerve, this gives off small branches. In the bulbar portion of the nerve run small vessels from the choroid, and often branches of the short ciliary arteries pass from the sclera into the nerve.

In a number of cases of embolism the cilio-retinal vessels are seen after a time to have established a capillary network on or near the disc. In cases in which there is from the beginning a small macular artery free, giving a small field near the point of fixation, some writers have supposed this vessel to be a cilio-retinal vessel, which probably in most cases it is not.

All of the recent text-books and monographs that treat particularly of this subject state that the re-establishment

FUNDUS ERECT, FIELD INVERTED IN ALL.

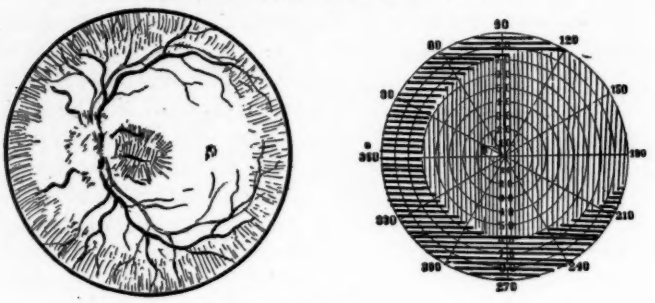


FIG. 1.

Embolism of central artery excepting macular branch (L).

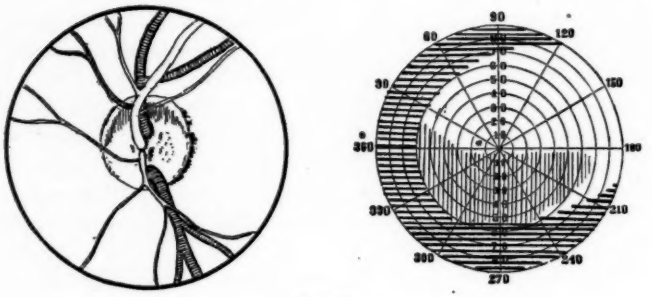


FIG. 2.

Embolism of inferior papillary artery from which median artery is given off (L).

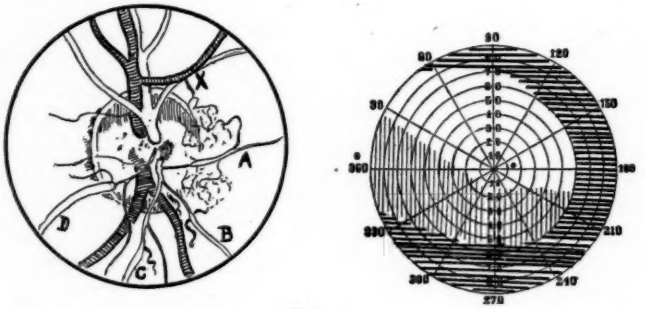


FIG. 3.

Embolism of inferior papillary artery; collateral restoration of circulation in each branch (R).

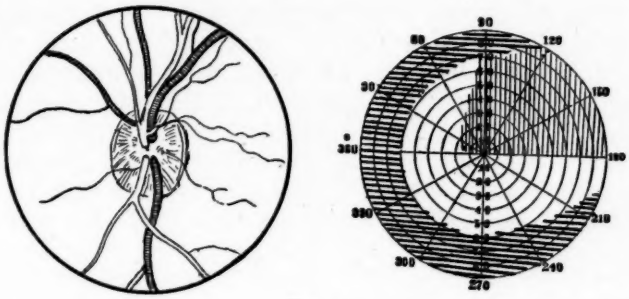


FIG. 4.
Embolism of macular branch from superior papillary artery (L).

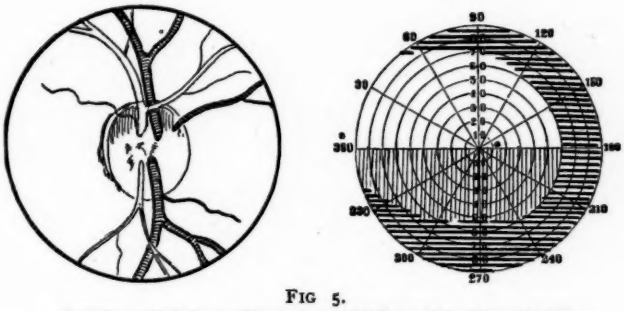


FIG 5.
Embolism of inferior papillary artery which has no lateral branches (R).

of the circulation occurs by the lumen of blocked vessels again becoming free.

Michel in his text-book says: "The re-establishment of the circulation does not occur by collateral vessels or by the so-called cilio-retinal vessels present. It is rather to be assumed that if the embolism has not blocked the artery incompletely at first, the channel soon becomes at least partially free." Noyes in his text-book speaks in a somewhat similar manner. Nettleship (text-book) says: "Sometimes the pressure test, which showed blockage of some or all branches shortly after the onset, again produces pulsation a few days later without visible evidence of collateral circulation, thus proving the re-establishment of the main channel." Nettleship, however, in the *Helmholtz Festschrift* last year, reported a case of embolism of the main artery in which there was evident anastomosis between ciliary and retinal vessels.

Our Case 3 is a very remarkable example of such anastomosis. Here there are four divisions of the inferior branch of the central artery. Each branch is small or almost obliterated near its origin, and three of the branches may be seen to receive each an anomalous anastomosing vessel, after which it nearly or quite regains its normal calibre.

In the centre of the disc (Fig. 3) is a white mass which would seem to be the remains of a blocked vessel.

From the superior nasal artery *X*, a small twig is given off which breaks up into a capillary network. This network is joined by a superior twig of an inferior nasal artery *A*, which in the papillary portion of its course is reduced to a mere thread. An inferior twig from the artery *A* joins a second capillary network below. Apparently anastomosing with this network is a dark tortuous vessel which joins the inferior artery *B*. At a corresponding point on the opposite side, *B* is joined by a second dark tortuous vessel. Centrally from the junction of these two ciliary vessels the artery *B* is a mere thread.

Artery *C* shows a blood column but is small up to a point a short distance below the disc, where it is joined by a tortuous vessel.

A small artery emerges by itself in the centre of the disc, and running inferiorly divides into two branches, one of which unites with the artery *D*. The artery *D* apparently emerges at the margin of the disc and is of normal size. It is difficult to say whether the small artery which emerges in the centre of the disc is to be considered the narrowed central portion of artery *D*, or whether it is a distinct vessel which gives off an anomalous anastomosing branch, joining artery *D*.

In the infero-nasal portion of the disc is another cilio-retinal vessel which has no apparent connection with the retinal vessels.

These cases show that, with a single embolism of a branch of the central retinal artery, we may have a field of quite irregular form, which to a considerable extent may be explained by the variations of arterial distribution; and that in cases where the lumen of an artery remains blocked, we may have a collateral restoration of its circulation by anastomosing vessels.

ON TRAUMATIC ENOPHTHALMUS.

By Dr. THEODOR BEER,[1] of Berne, Switzerland.

Translated by Dr. Ward A. Holden.

(*With one figure in the text.*)

CASES of traumatic enophthalmus, in which, after wounds or contusions in the region of the eye, the globe sinks into the orbit, are so rarely observed and have been explained in so many different ways, that a description of every case seems called for.

The following cases have been reported:

1. Himly (*Krankheiten und Missbildungen des menschlichen Auges.*, 1843, Part 1, p. 395) saw a marked enophthalmus come on suddenly after a blow in the neighborhood of the eye. The eyelids were closed and sunken, apparently as if no globe was present. When the lids were separated a ball normal in size, appearance, mobility, and vision was found. He supposed that the pulley of the superior oblique muscle had been loosened and that therefore the tendon of this muscle could not draw the eye forward.

2. Nieden (*Klinische Monatsbl. für Augenheilk.*, xix., p. 72) saw enophthalmus in the right eye of a builder on whom a mass of earth had fallen from a distance of twelve feet. No wound was discovered. The ecchymotic right half of the face regained its normal color and form eight days later. Fourteen days after the injury he found that there was something wrong with his right eye.

[1] From Prof. Fuchs' Clinic in Vienna.

The orbital margin of the lids is marked by a sharp line, and the lids are retracted into the orbit. Both ball and lids have their normal mobility. The right ball lies about 4 *mm* deeper than the left.

Nieden explains the condition by assuming a disappearance of the orbital fat, in consequence of the long-continued and forcible compression by the mass of earth.

3. Talko (reviewed in *Klin. Monatsbl. für Augenheilk.*, xix., p. 471) saw a case in a soldier whose right eye was injured by a falling stick of wood. The sight at once began to fail and there was periodic pain above the eye.

Seven months after the injury, the right lids were retracted as in phthisis bulbi, and the palpebral fissure was decreased in size. When the upper lid was elevated the ball rolled upward. Pupil and fundus normal. In the right eye there was no vision, in the left fingers were counted at ten feet. A pulsating pain in the region of the eyes, temples, and ears, particularly at night. After some months there was complete blindness, while the pain and retraction of the ball remained as before. The right globe lay six lines behind the superior margin of the orbit, the left four and one half lines.

Talko assumes an anomaly of the trophic or vaso-motor ocular nerves, which would be indicated by the retraction of the ball, the disappearance of the orbital fat, the over-filling of the eye with blood, and the late pathological changes in the cornea.

4. Gessner (these ARCHIVES, xviii., p. 269) saw three cases. The first was a miner who was struck by falling coal and lay unconscious. He sustained a fracture of the right inferior maxilla and zygoma, and also an injury of the upper margin of the orbit. When after ten days the swelling of the lids was less and the right eye could be opened it was remarked that the right eye was smaller than the left.

At the outer two-thirds of the superior orbital margin there is a non-adherent cicatrix. The lids are sunken and the palpebral fissure is 2 *mm* shorter than in the other eye. The function of the levator is disturbed, and the mobility of the ball upward is limited. Motion downward is exaggerated, in the other directions

normal in extent but sluggish. The right eye lies 3 *mm* deeper than the left. V = $\frac{3}{4}$-$\frac{3}{5}$. No diplopia. Pressure in the trochlear region causes intense pain.

In the second case a large lump of coal had fallen on a miner's forehead. Fourteen days later when the right eye was opened, it appeared smaller than the left. There is a non-adherent cicatrix at the outer two-thirds of the superior orbital margin. The upper lid is sunken. Movement of lids normal; upward movement of ball limited. Ball lies 2–3 *mm* deeper than the other. V = $\frac{1}{4}$. The amblyopia may have existed previously. Pressure into the orbit elicits pain.

The third case was a miner who was thrown down by a falling piece of coal. Right eye swollen. When it was opened fourteen days later he noticed that it was smaller than the left. There was an adherent cicatrix at the nasal third of the superior orbital margin. The upper lid is deeply sunken, and cannot be raised. Limited movement of the ball upward and outward, increased downward. Homonymous diplopia when he looks to the right. V= $\frac{1}{3}$. Ball 3–4 *mm* deeper than the other.

Gessner does not deny the possible truth of Nieden's theory of trophic disturbances in the orbital tissues, but finds a more probable cause in the orbital injury which all his cases showed. He assumes a cicatricial contraction of the retrobulbar cellular tissue following inflammation and periostitis due to the injury. With the shrinking of the tissues behind it the eyeball simply sinks back into the orbit.

5. Von Becker of Helsingfors (*Arch. für Ophth.*, xii., p. 289) reported the following case: Three months previously a peasant woman was struck in the left eye by a cow's horn so severely that she lay unconscious for four days. Blood flowed from the wound and from the nose. The left cheek was swollen and ecchymotic, sensibility almost wanting. The vision of the right eye failed.

St. pr.: At the supra-orbital margin is a non-adherent cicatrix extending from a point above the outer commissure, 25 *mm* inward. The upper lid is sunken. Beneath the supra-orbital margin is a white cicatrix in the conjunctiva. The finger may be carried 20 *mm* deep into the orbit before any resistance is encountered. The upper margin of the cornea lies two lines above the infra-orbital margin. The eye is normal. Fingers can

be counted in the upper portion of the field. Ball immovable. Slight paralysis of left facial muscles. Left upper teeth somewhat sensitive. There is no foreign body producing the displacement of the ball, which is adherent to the infero-nasal wall of the orbit.

According to von Becker the cow's horn passed into the outer third of the superior portion of the orbit. The conjunctiva was torn. The ball when struck behind the equator rolled downward and inward, and was pressed forward. The horn fractured the lachrymal bone, the lamina papyracea, and perhaps also a portion of the roof of the antrum. Some of the soft parts healed in the cavity so made, and attached the ball in its present position. The branch of the oculo-motor supplying the levator is unaffected, the other branches may be torn as there is no mobility of the ball.

6. Lang (*Trans. Ophth. Soc.*, vol. iv., p. 41) saw the following case: A boy of thirteen was knocked down in the street by a wagon and received a cut in the right brow, which was sutured. For twelve hours he had severe hemorrhage from the nose. When the swelling of the lids passed off, the right eye was seen to be sunken in the orbit. One month later there was a non-adherent cicatrix in the outer two-thirds of the brow. The palpebral fissure was 4 *mm* shorter than the left. Mobility of the eye diminished in every direction, particularly inward. The ball was dislocated slightly downward. The right cornea lay 8 *mm* behind the level of the left. Doubtful anæsthesia of the right cheek and forehead. No apparent alteration in the bony wall of the orbit. No pain on pressure. Lang's probable explanation is that the trauma caused a fracture or depression of a portion of the orbital wall, and the orbital fat is not sufficient in quantity to fill the enlarged post-ocular cavity, so that the ball sinks in from atmospheric pressure.

7. Tweedy (*Arch. für Ophth.*, xiii., 2, p. 457) saw a case similar to that of von Becker. The horn of a cow caused a perforation of the floor of the orbit which passed into the antrum. The eye being blind was excised. There were numerous adhesions. The fracture of the superior maxilla must have been very extensive, as for a time the ability to chew on the injured side was lost, and the teeth were very painful.

8. Langenbeck (*Arch. für Ophth.*, xiii., 2, 457) saw a similar case. A railway workman was struck on the right half of the

face by a locomotive. The upper lid was torn vertically and horizontally, and detached from the orbital margin. A deep wound extends vertically from the inner angle of the eye through the cheek to the upper lip. Through this wound the sound passes into the antrum. No eyeball can be found in the orbit. In its place is a bluish-black, soft, pulsating vesicle. It was supposed that the greater portion of the eye had been torn out, and that the blue pulsating mass represented the choroid. When the swelling had grown less eight days later, the ball was seen to be projecting through a defect in the floor of the orbit into the antrum. When replaced in position the ball was found to be uninjured and the vision normal. Four months later an ulcer appeared in the cornea, which finally led to phthisis bulbi.

9. Nagel (cited in *Graefe-Saemisch*, vol. vi., p. 507) observed a case in which the root of the nose and the inner half of the left lower orbital margin had been injured by the kick of a horse, and enophthalmus had developed. The ball was depressed 3 *mm* and sunken 2–3 *mm*.

10. Schapringer (*New York Medic. Monatsschr.*, June, 1890). A girl of seven fell against an iron grating. She did not lose consciousness but was very dizzy. No hemorrhage from nose or ear. Two hours later her mother noticed a drooping of the right upper lid and a retraction of the ball. The right cheek was ecchymotic.

The right upper lid droops covering more than half the pupil. The lid rises with the cornea in upward vision. The right eye lies 2 *mm* deeper than the left. The tension of the right eye is decreased. Motility normal. No disturbances of sensibility. Schapringer assumes, in explanation of the three symptoms, ptosis, enophthalmus, and hypotony, an affection of the sympathetic nerve due to bruising or hemorrhage. Three days after the injury, all the symptoms had disappeared.

11. Löw (Inaug. Dissert., Berlin, 1890). A man of forty-three was kicked in the left eye by a horse, and lay unconscious for two days. Six days later the lids could be opened and he discovered that he could not see. A sensitive scar extended from the inner angle of the lids upward to the orbital margin. The left eye lay 6 *mm* deeper in the orbit than the right. The infra-orbital margin showed a deep depression at the junction of the middle and outer thirds. The mobility of the ball was normal inward, restricted upward and downward, and wanting outward.

The optic nerve was atrophic. Löw supposed that there had been a cicatricial contraction of the orbital cellular tissue and probably a partial cicatricial adhesion of the ball.

12. The first of my cases was seen in Prof. Fuchs' clinic. A man of forty-two was kicked in the right eye by a horse. He fell unconscious, bled, and vomited, but two hours later came to. A surgeon sutured two wounds. Eight days later the swelling was less, and when the eye was opened the patient found that

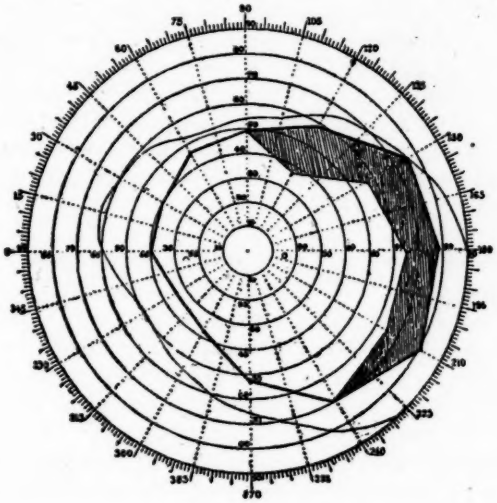


FIG. 1.

Field for white; shaded portion represents a zone of diminished visual acuity.

he could not see well with it. There was no pain, but a "woolly feeling" in the eye. The retraction of the ball was noticeable ten to twelve days after the injury. Five months later when examined a depressed but not adherent cicatrix 3 *cm* long was seen running obliquely through the brow at the supra-orbital foramen. There was a corresponding bony defect. 3 *mm* outward from the outer commissure a linear non-adherent cicatrix 12 *mm* long runs downward and outward. At the upper end of this cicatrix is a small shallow depression in the orbital margin. The finger could be passed deep into the orbit and no evidence

of fracture or depression was found. No portion of the orbital wall was sensitive to pressure.

There is slight ptosis. The palpebral fissure is 3 *mm* narrower than the left, and 4 *mm* shorter. The right ball lies 7.4 *mm* deeper than the left, and 2 *mm* lower. Elevation of the ball and lid restricted. Iridodialysis upward and outward, pupil irresponsive. V = $\frac{6}{36}$ not improved. Probably paralysis of accommodation. Patient could not read. The disc showed a post-neuritic atrophy. Visual field and color fields show a moderate concentric contraction. To the temporal side a relative scotoma.

13. My second case was a fifteen-year-old Alpine guide, who a month before had fallen ten feet, striking the left side of the face against a large stone. He did not become unconscious. Eight days later retraction of the left eye was noticed. No pain, vision normal. The left eye lies 3 *mm* deeper than the right, the upper lid droops but can be promptly elevated. No restriction of mobility; no diplopia.

If we now arrange these thirteen cases in groups according to the degree of enophthalmus, the case of Schapringer will come first. Here there was a transitory enophthalmus of 2 *mm* appearing two hours after the injury, hypotony, and sympathetic ptosis. The enophthalmus according to Schapringer was due to a paralysis of Müller's orbital muscle from a lesion of the sympathetic.

There is no doubt but that a vascular spasm in the retrobulbar tissue, by decreasing the bulk of this tissue, may cause a transitory enophthalmus of varying degree. A traumatic irritation of the sympathetic vaso-motor fibres in the retrobulbar tissue would explain the slight and transitory enophthalmus in Schapringer's case. The hypotony is in accord with this explanation, and the ptosis may be considered a purely mechanical consequence of the enophthalmus.

In the second group would be the cases of Himly, Nieden, Gessner, Nagel, my two cases, and with some reserve the cases of Lang and Löw also. In these cases, some time after a more or less intense contusion of the ocular region, a considerable degree of enophthalmus was observed, which was permanent.

The cause of the enophthalmus, surely in most of these cases and probably in all, is an atrophy of the retrobulbar cellular tissue. Cicatricial contraction of the retrobulbar tissue, paralysis of the orbital muscle, and fracture of the trochlea, are not rational explanations. In Nieden's case we have a neurotic atrophy of the cellular tissue, and also in my first case. Here the symptoms were rupture of the skin, injury of the orbital wall, with corresponding non-adherent cicatrix. No marked pain, excessive enophthalmus after twelve days, ptosis, restricted mobility upward. $V = \frac{3}{\infty}$, neuro-retinitis, and paræsthesia which also indicates a trophic neurotic disturbance.

Gessner's three cases cannot be explained as having been caused by orbital periostitis and phlegmon.

Löw's case was not due to cicatricial contraction of the cellular tissue, but to cicatricial adhesions of the ball itself.

While it is fairly certain that the enophthalmus coming on shortly after contusions in the region of the eye is due to diminution in the bulk of the orbital tissues from atrophy, the causal connection between this affection and the trauma is not clear. According to analogy we should suppose the atrophy of the orbital tissues to be due to a lesion of a nerve centre or tract, and particularly of the sympathetic or of the trigeminus. In neurotic atrophy of half of the face we have a picture similar to that seen in traumatic enophthalmus.

Further observations will be required to prove the correctness of my theory of traumatic enophthalmus. Even if in some cases there be found a cicatricial retraction of the ball or a depression in the orbital wall, the theory may still account for the majority of the cases.

After the manuscript of this paper had left my hands, a paper appeared by E. Heese "On the Influence of the Sympathetic on the Eye," etc., (*Archiv für die gesammte Physiologie*), vol. 52, p. 535, in which is given the experimental proof of my views. The following extracts are of interest.

"In the rabbit, irritation of the sympathetic produces a retraction of the eye. Cutting the sympathetic leads to a slight protrusion of the ball."

"In the cat and dog immediately after irritation there is a marked protrusion followed by retraction."

"In the newly killed rabbit the contrary condition to that observed in life is seen. Sympathetic irritation causes a slight protrusion. The different conditions of vascular tonus and the corresponding varying amount of blood in the orbit must influence the position of the ball."

"There are two forces governing the position of the ball; one is the state of contraction of the vessels with the consequent varying amount of blood in the orbit, and the other is the condition of the musculus orbitalis."

THE MEDICO-ELECTRIC EYE-BATH IN THE TREATMENT OF SCLERITIS AND EPISCLERITIS.

By Dr. JOSEPH NORSA, of Rome.

Translated by Dr. J. M. Mills, of New York.

SCLERITIS and episcleritis are found to be not infrequent accompaniments of gout and rheumatism, as well as of syphilis.

The chronic nature of this disease is well known, as is also the fact that, even with the most attentive and rational treatment, only a partial or temporary improvement is obtained, as the disease has a tendency to relapse, particularly in the part first affected, and also readily extends to the rest of the membrane.

It has been observed, in most instances, that the movement of the muscles of the eye is extremely painful. The inflammation of the sclera is indicated by sensitiveness to light, tearing, swelling of the tissues, and considerable capillary injection. The cells of the sclera become infiltrated, thereby causing the development of the capillary vessels, which serve, to a certain extent, to hasten the absorption of the exudation (Galezowski). The sequel, at times, is atrophy of the physiological elements, with more or less thinning of the sclera. Though the inflammation itself may not be severe, still at times it produces complications in the other membranes of the eye, such as keratitis, iritis, or choroiditis. The latter secondary affections are usually observed in syphilitic, scrofulous, or cachectic individuals.

As regards the treatment of this disease, improvement has been obtained from the use of atropine or cocaine or fomentations with lukewarm aromatic water, by scarification of the episcleral swelling (Adamück), or by deep incisions into the sclera (Darier), by scraping of the diseased scleral tissue, but especially by applying the steam-douche, for twenty minutes, two or three times daily, by massage of the eye if the healing is sluggish (Pagenstecher), and by injection of pilocarpin (De Wecker), and of sublimate solution (1:5000) into the scleral tissue (Snellen).[1]

At the same time it is, of course, necessary to prescribe internal treatment adapted to the general malady, of which the scleral affection is only a local manifestation. Therefore mercury and the iodine preparations are beneficial in episcleritis of syphilitic origin, whereas sulphate of quinine, colchicum, salicylate of lithium (Dufour, Panas[2]), and diaphoretics or mineral waters containing iodine, are useful in rheumatic and gouty scleritis and episcleritis. Frequently, however, these remedies are unable to limit the duration of the disease or to prevent a relapse. The cause of this, in my opinion, is that the local application is too superficial, and consequently only produces a transitory effect upon the membrane; and also that the internal treatment, entrusted to the circulation, operates only imperfectly and does not always reach the seat of the disease in the eye.

In order to overcome the difficulties previously met with in the treatment of this disease, I have endeavored to avail myself of past experience (in which, however, the internal treatment acted too slowly and imperfectly), by effecting a direct absorption of the medicament into the scleral tissue. This end has been attained by employing a remedy very useful in many other eye affections, namely, electricity. Acting upon the theory that the galvanic current has cataphoric power, and, at the same time, assists the absorption of the exudation by means of its catalytic properties, we made a number of experiments in severe, mild, and relapsing scleritis and episcleritis, in order to ascertain if this current,

[1] Prof. Snellen, *Ophth. Soc. Un. Kingdom*, Nov. 14, 1889.
[2] *Congrès d'Ophtalmologie de Paris*, 1888.

by transferring the proper medicated solution to the eye, could not effect a permanent and quicker improvement than could previously be obtained by the usual therapeutic measures. The method chosen for applying the electric treatment was the lukewarm eye-bath and the fluid was a 1–2 per cent. solution of salicylate of lithium. We were induced to try this treatment by the brilliant results of Dr. Denti[1] (and also my own[2]) in the treatment of other eye affections, particularly of the optic nerve, by means of the simple hydro-electric bath. We mention three cases, from among the many treated, as they had been of long duration and had resisted all other therapeutic measures, and therefore afforded a good opportunity of testing the efficiency of the treatment.

The method of using the eye-bath and the manner in which it works are as follows: A two-litre glass vessel, filled with a lukewarm 1–2 per cent. solution of salicylate of lithium, is placed two metres above the head of the patient. This reservoir communicates, by means of a rubber tube, with an ordinary eye-glass; at the bottom there is a perforation, through which a metal tube is passed. The outer end of this is fastened to the rubber tube, the inner end projects about ½ centimetre above the opening of the eye-glass. A small stop-cock placed on the metal pipe serves to connect or disconnect, as desired, with the water reservoir. There is also an opening in the side of the vessel provided with a stop-cock, in order to let off the water. Then, finally, we require an electric apparatus, preferably with a galvanometer, for developing a constant current; also a myriamperometer is desirable in order to regulate the exact strength of the current. One electrode communicating with the metallic plate, properly covered and dampened with salt water, is laid upon the cervical sympathetic, while the other is connected with a small screw on the metal covering of the eye-glass. It is immaterial whether the positive or the negative pole is placed on the eye or glass. This can be regulated by the sensitiveness of the

[1] Dr. Denti, *Saggio di Idro-Elettro-Terapia Oculare*, Milano, 1885.
[2] Dr. Norsa, *Un Biennio di Clinica Oculistica*, Roma, 1889.

patient to the one or the other. The electric bath is applied to each eye for about five minutes, and it is unnecessary to instil cocaine beforehand, as even nervous and feeble patients can bear it easily. This treatment can be repeated every day, as the hyperæmia of the conjunctiva, and especially that of the ball, after this treatment disappears, at the longest, in two hours. The patient experiences only a mild and easily borne sticking sensation of short duration.

[The report of three cases cured by the described procedure is omitted in the translation, for want of space. H. K.]

FURTHER COMMUNICATION ON THE TREATMENT OF TRACHOMA.

By H. KNAPP.

(*With one wood-cut.*)

IN the January number of 1892 of these ARCHIVES (vol. xxi. p. 119) I published my experience in the treatment of trachoma with the roller forceps, based on the observation of 114 successive cases. Since that time this treatment has been continued regularly in all suitable cases not only by myself, but by my associates at the N. Y. Ophthalmic and Aural Institute and the Eye Department of the Vanderbilt Clinic. Being familiar with this mode of treatment and its action and results, we have not so carefully recorded the cases as in the first series. Many of the ordinary follicular, uncomplicated kind, which, as usual, were promptly and permanently cured by one operation and a short after-treatment, have not been entered into the record book, but no cases were omitted that showed any abnormity either in their primary condition or in the course of healing, in particular such as were followed by relapses. After the first series (of 114 cases) from March to December 16, 1891, a second series of 86 cases (from the end of December, 1891, to the middle of January, 1893) is recorded. I now propose to report on these as well as on the relapses which occurred in the first series.

The *mode of operation* has remained the same as I described it in the above communication. After many trials Geo. Tiemann & Co. have furnished us with a satis-

factory instrument which we have used for the last year. (See accompanying drawing.)

By a singular mistake, for which I cannot account, the length of the rolling cylinder has been misstated in my publication as being 20 to 25 *mm*. It never was more than 9 or 10. The mistake in the former publication is evident if we consider that 25 *mm* is one inch, which would make the instrument much too large. This error, which has also crept into the German translation of my paper, came to my notice only a few days ago when I read the article of Sydney Stephenson on "The Surgical Treatment of Trachoma" in the January number of the *Ophthalmic Review* (vol. xii., p. 2), where he says that he has modified the roller forceps in making the cylinder only 9–10 *mm* long, but 3 *mm* thick instead of 1–1.5 *mm*. In the instruments which I have used during the last year the cylinder is 2 to 2.50 *mm* thick. These cylinders turn very easily, and I do not doubt that those of 3 *mm* thickness turn still more readily, for the longer the lever the easier the revolution. Yet the thickness of the roller has its limits, for the bar has to be introduced between the lid and the eyeball, and the cornea should not be scratched.

In the new instruments the cylinder has a pivot at each end, fitting into a perforation at the ends of the legs of the stirrup. These legs are longer than in the former model and can be pulled apart so that the cylinder may be removed, cleansed, and sterilized after each operation. The creases in the roller have remained the same. Before use the roller is held into boiling water. I have never seen the cornea injured by the action of the roller, nor have I noticed any kind of infection from it. The instrument makers tell me that the demand for the forceps has been very large and steady, and almost no complaint has come to their notice.

In all severer cases of trachoma I use general anæsthesia, for the operation is tedious and *the success depends on its*

thoroughness. All the soft trachomatous material should be pressed out from every portion of the conjunctiva in which it is deposited, visible or invisible. This can be done at the commissures and caruncle as well as in the tarsal and retro-tarsal portions. The roller must pass repeatedly over each place to secure the complete liberation of the trachoma substance. This substance being soft and gelatinous, it will come out by a pressure that does not interfere with the integrity of the cellular and fibrous elements of the conjunctiva. There is commonly free hemorrhage, which seems to be rather an advantage than a drawback as to the recovery. In no case have I seen sloughing or ulceration of the conjunctiva. Small ruptures of the surface of the conjunctiva occur, of course, whether we scarify this membrane or not. In the next days the posterior part of the palpebral conjunctiva is agglutinated to the reflex fold in many cases, and the advice is given to solve these adhesions with a probe. I have done this also at the beginning, but have found it of no importance, for these agglutinations disappear with the swelling and the fibrinous deposit of the conjunctiva. Where portions were cut off, adhesions and shortening were permanent.

The *indications* for the expression treatment are contained in the following sentence: *Expression is indicated only where trachomatous substance can be pressed out.*

In the *first place* are those cases in which spawnlike granulations are deposited in the conjunctiva without inflammatory irritation, the cases of *pure, non-inflammatory, follicular trachoma.* They are frequent in New York, but seem to be rare in other places, as would appear from a case observed at the University Eye Clinic of Berlin, described and depicted by Dr. R. Greeff, under the name of pseudo-trachoma, which ended in spontaneous recovery. Dr. Greeff's description is identical with what I see in New York every week. This variety of trachoma is not infrequently limited to one eye, and I doubt very much whether it is contagious. I am inclined to consider it as an analogue of the adenoid or follicular hypertrophies of the naso-pharynx. They occur, as the latter, mostly in youth, rarely under five

years or over twenty. I have no experience of their spontaneous disappearance without producing atrophy of the conjunctiva, but do not doubt it. Expression cures the great majority of them without any other treatment, but it alone does not cure them all. A small number return with relapses, usually mild ones, which are speedily cured by another expression. A few are made worse by expression, *i. e.*, the apparently pure, non-irritative, follicular trachoma is, by the mechanical treatment, converted into the inflammatory form of trachoma. The mucous membrane swells, burns, pains, secretes, and in the course of weeks becomes again the seat of miliary or spawnlike granulations, just as we see this deposition occur in the course of acute catarrhal conjunctivitis. I have noticed five such cases. The primary treatment of this inordinate reaction should be—as in the period of reaction in every case of expression—purely antiphlogistic: bathing the lids with cold water and washing the conjunctival sac out with a mild antiseptic solution. When the reaction is over, and the conjunctiva remains swollen and uneven, the so-called papillary hypertrophy, nitrate of silver (one per cent. solution) is of advantage, and when the spawnlike granulations have developed, and the conjunctiva is not much swollen, the rolling should be repeated. Of late, I have mostly used the sulphate of copper crystal as after-treatment, until the conjunctiva was smooth and completely free from granulations.

The *second class of trachoma*, which may be designated as *secondary, consecutive, inflammatory, or complicated trachoma*, comprises those cases in which the deposition of granules is preceded, accompanied, or succeeded by some form of conjunctivitis, commonly catarrhal or blennorrhoic in its nature. These cases *are contagious* (perhaps from the microbes of the original or complicating conjunctivitis) and *destructive*, producing shrinkage of the conjunctiva and corneal ulceration. Mostly both eyes are affected. This vast class of cases, which are chronic, with a marked tendency to relapses, is not cured by expression alone, yet it is greatly benefited by it. As long as there is hyaline infiltration, either granular or diffuse, this should be pressed out. I scarify such

eyelids rather deeply, press the liquid and soft material out, and rub a solution of bichloride of mercury, 1:500, into the tissue with a pellet of absorbent cotton, or a tooth-brush, the grattage of Darier and others. This treatment is based on mechanical and germicidal principles, according to generally accepted theories. The results have, of course, not been so brilliant as in the non-inflammatory, follicular trachoma, yet the treatment has abridged the recovery essentially, and in many cases, I think, also set a stop to the progressive destruction of conjunctival and corneal tissue. This surely is an immense advantage. I have not seen that corneal ulceration or pannus has been made worse by this kind of treatment. It is not infrequent to see the ulcerous or pannous cornea become somewhat more opaque during the period of reaction from the mechanical and caustic treatment, but the increase of the opacity disappeared quickly in all cases that have come under my notice, and the corneæ afterward became clearer than they were before the operation.

A *third form* of trachomatous disease, which I have seen on the upper lids only, and I would call *sclerosing trachoma*, are those tough excrescences which are most obstinate and when once fully developed resist all kinds of treatment. Cases which I have watched from the beginning show in their initial stage the ordinary, rather vascular, lid granules. The copper crystal does not affect them, or rather favors their growth. In the course of months and years they multiply and cover the upper tarsal and retrotarsal conjunctiva in the shape of irregular, hard, red excrescences, flattened on their surface by the pressure of the thickened and heavy lid. Some of them can be raised with a probe, when they exhibit a pedicle and a mushroom-like, often notched, and lobulated head. Cut off with scissors they feel tough, and show under the microscope an irregular structure of dense fibrous tissue surrounded with vascular epithelium. In their further course they become pale, and ultimately completely white. The everted lid looks like a section of a dense fibroma. The disease lasts years, has no great tendency to affect the cornea, resembles in a certain stage the pale, tessel-

lated tarsal conjunctiva of Saemisch's spring catarrh, but never shows the circumcorneal thickening. Strong remedies make it worse; mild washes, in particular a 1:5000 solution of bichloride of mercury brushed over the inner surface of the upper lid five or six times daily, seem to do good. The disease may be, and as far as I know usually is, recovered from without great roughness of the conjunctiva or damage to the cornea.

I classify this affection among the trachoma group because its beginning, when I had an opportunity to observe it, is distinctly that of follicular trachoma. Its course, however, is characterized by an inordinate development of tough fibrous tissue, a fibrous degeneration of the granular substance. It is distinguished from the cicatricial form of trachoma by the want of atrophy. The tarsus, instead of being shrunken and curved, is thickened and hypertrophied, a regular keloid scar in appearance and microscopic structure. When once this fibrous degeneration has taken place, expression and grattage are of no service, if I may judge from the observation of a few cases, but expression is decidedly beneficial in the first stage. This has been demonstrated by a recent case which on account of its importance I beg leave here to detail.

Robert L., a seventeen-year-old lad, of Hoboken, came to me complaining of swelling and drooping of his left upper eyelid. He had no pain, no discharge, no discomfort in the use of his eye. The right eye was normal. On everting the left upper lid, the tarsal and retrotarsal portions were red and swollen. The retrotarsal fold in its inner two thirds was densely beset with small spawnlike granules, whereas the outer third showed several red, and irregular excrescences which projected over the adjacent tarsal conjunctiva. The whole tarsal conjunctiva was red and uneven. The lower lid was intact. I considered the case one of simple follicular trachoma in transition to become sclerotic. I cut the excrescences off and put them in Müller's fluid, and then pressed out the granulated and diffuse hyaline substance from the nasal side of the lid. The next day the inner surface of the lid was red and swollen, but perfectly smooth, and the patient felt well. The day after, there was more swelling, but

no discomfort. On the third day there was swelling of the lid and conjunctiva, heat, pain, lachrymation, but no mucous or purulent secretion. The fourth day, the tarsal conjunctiva showed a number of yellowish, miliary infiltrations. The other symptoms the same. Cornea intact. I let him bathe the eye with cold water as often as this felt grateful. Then the swelling began to subside; I ordered bichloride of mercury 1:6000 to be brushed into the eye four times daily. The miliary infiltration disappeared without a trace, the swelling of the conjunctiva passed away, and four weeks after the operation the eye was as widely open as the other, the upper lid no longer swollen; the inner surface, though still redder than normal, was perfectly smooth, without a trace of granulation. Where the excrescences had been cut off, there was a slight scar to which the posterior portion of the conjunctiva adhered.

In cases of cicatricial trachoma, *i. e.*, the end stage of the uncured cases, expression, or any other form of mechanical treatment, is no longer indicated. When there still is some swelling and a partial infiltration of the lids, scarification and impregnation of the conjunctiva and tarsus with stronger solutions of bichloride may be resorted to in order to terminate more quickly this excessively tedious disease.

Summing up the results of the treatment sketched above, they are as follows:

1. Rapid, perfect, and permanent recovery by expression alone, or expression followed by a short course of mild caustic treatment, takes place in the majority of cases. This refers chiefly to the cases of pure follicular trachoma.

2. Imperfect recovery, *i. e.*, disappearance of trachoma, but leaving more or less shrinkage of the conjunctiva, is the common issue of old, neglected cases of inflammatory trachoma.

3. Relapses occur of both simple and inflammatory trachoma. They have been recorded in ten cases of the first series, and in eight of the second. The majority of patients come back with a relapse after having felt comfortable and done nothing for their eyes many months after the operation. The relapses were mostly promptly cured by a second

expression; in a few cases a third was necessary. In two cases the steady after-treatment with the copper crystal did not prevent a relapse. They had been treated as out-patients and worked in a dusty atmosphere.

4. The operation itself has neither destroyed nor injured an eye.

In conclusion I may say that the mechanical treatment, with or without scarification and impregnation of the lid with corrosive sublimate, does not cure all cases of trachoma, but it benefits and cures the great majority, and always abridges the recovery. In rare instances it converts a simple trachoma temporarily into an inflammatory trachoma, but injures no case permanently.

A REPORT OF THE PROCEEDINGS OF THE TWENTY-SECOND MEETING OF THE HEIDELBERG OPHTHALMOLOGICAL SOCIETY, HELD AUGUST 8, 9, AND 10, 1892.

By Prof. KARL HORSTMANN.

Translated by Charles H. May, M.D., New York.

1. Uhthoff (Marburg).—*The Ocular Disturbances of Syphilis of the Brain.*

During the past nine or ten years, Uhthoff observed 100 cases of syphilis of the brain; autopsies were made in seventeen of these cases. Changes were found not only in the eyeballs, chiasma, and optic tracts, but also in the nuclei. The eyeballs presented gummous iritis, irido-choroiditis, and changes in the retinal blood-vessels; the optic nerves showed more or less interstitial neuritis with perineuritis and localized gummata. When the latter occur at the chiasma, symptoms of temporal hemianopsia are developed—Uhthoff saw such a case. Another case presented complete obliteration of the ophthalmic artery, producing unilateral optic-nerve atrophy. Among the ocular nerves, the motor oculi was affected most frequently—six times unilaterally, twice bilaterally. In two cases there was paralysis of the abducens, in three cases paralysis of the trigeminus, in two cases disturbances of smell, and in two cases disturbances of hearing.

Michel (Würzburg) and Sattler (Leipzig) took part in the discussion; the former believes that the vascular changes are primary in most cases.

2. Schirmer (Königsberg).—*Sympathetic Inflammation without Perforation of the Globe of the Eye Primarily Affected.*

Schirmer gave an abstract of a paper about to be published on a clinical study of the etiology of sympathetic inflammation fol-

lowing intraocular tumors and subconjunctival ruptures of the globe. Out of thirty cases found in literature, in which various authors attribute sympathetic ophthalmia to the existence of a tumor, twenty-seven do not stand the test of severe criticism ; in the other three cases there was marked irido-cyclitis in addition to sarcoma of the choroid. On this account, the reader of the paper attributes the sympathetic ophthalmia to the irido-cyclitis, and not to the sarcoma, and demonstrates that the inflammation cannot be caused by the tumor—the former being merely an accidental, genuine irido-cyclitis, or one produced by endogenous infection ; in both cases we should be very guarded in making the diagnosis of sympathetic ophthalmia, and we certainly ought not to consider the tumor as the primary cause. Twenty cases had been reported in which sympathetic inflammation was reported as following subconjunctival rupture of the globe ; only five of these were worthy of consideration. In all of the cases there was a severe irido-cyclitis, which could not have been the direct result of contusion, since healthy tissue will not become involved in progressive inflammation after a simple bruise. On the other hand, the form and the course of these cases were analogous to what is seen in infectious uveitis after perforating wounds. Schirmer considers this to be the etiology in these cases, and believes that microbes enter the eyeball either through minute lesions in the sclera, or that endogenous infection exists. The correctness of the Leber-Deutschmann theory and the improbability of the theory of purely nervous transmission are each strengthened by the fact that traction upon the ciliary nerves, so often seen, is not sufficient to cause sympathetic inflammation, the addition of infectious neuritis being necessary. He does not believe that the micrococci pyogenes are the active microbes, but thinks there are other unknown and hitherto undemonstrated species.

3. R. GREEFF (Marburg).—*Investigations in Sympathetic Ophthalmia.*

Greeff gives the bacteriological examinations of nineteen specimens of optic nerves which had been exsected, partly on account of the occurrence of sympathetic inflammation, partly because such inflammation had been feared. It was impossible to demonstrate the existence of microbes in the nerve or its sheaths by the various methods of culture, by anærobic breeding, or by inoculation of the anterior chamber of rabbits' eyes. Experi-

ments were also made by inoculating the vitreous of rabbits' eyes with putrid fluids, aspergillus fumigatus, staphylococcus pyogenes aureus, etc. In no case could transmission of microbes from one eye to the other by means of the optic nerve or its sheaths be demonstrated. It was only when general infection had occurred that microbes were found in the nerve or sheaths of the opposite eye,—in such cases the microbes having travelled by vascular routes. He claims that Deutschmann's experiments have not been confirmed, and hence Deutschmann's theory of migration cannot be regarded as proven.

In the discussion, Pflüger (Bern) remarked that the occurrence of sympathetic affections was accompanied by febrile manifestations. Schmidt-Rimpler (Göttingen) advised neurotomy of the optic and ciliary nerves, including the branches, to the insertions of the muscles. Uhthoff (Marburg) never found the same causative micro-organisms in bacteriological investigations of sympathetic ophthalmia. Leber (Heidelberg) thought the parasitic theory very probable, even though not yet proven.

4. Fischer (Dortmund).—*The Early Manifestations of Anchylostoma Duodenale in German Miners, and the Effect upon the Eye of the Anæmia Caused by the Parasite.*

The anchylostoma occurs in the intestine by hundreds; it fastens itself upon the walls and causes marked anæmia. The parasite does not occur in the eye, but retinal hemorrhages are observed as a result of the anæmia.

Samelsohn (Cologne) and Nieden (Bochum) took part in the discussion.

5. C. Hess (Leipzig).—*Filamentous Keratitis.*

There had been two theories concerning the nature of corneal filaments: the first regarded them as coagulation of fibrin from the cornea; the second, as coagula from the conjunctival sac. Hess demonstrates that the filaments consist essentially of more or less degenerated corneal epithelium. The disposition to the formation of these filaments is explained by a peculiar degenerative process in the epithelium; this process, which was partly unknown before, Hess describes more minutely.

Landolt (Paris) and Wicherkiewicz (Posen) observed filamentous keratitis in decrepit individuals. Samelsohn believes there are no cases of typical filamentous keratitis, regarding the filaments merely as accompanying manifestations. Leber opposes this view, and attributes the formation of filaments to rapid prolifera-

tion of epithelium; in this he was supported by Wagenmann (Heidelberg) and Sattler (Leipzig).

6. VALUDE (Paris).—*Contribution to the Study of Hemorrhagic Glaucoma.*

Valude examined four cases of hemorrhagic glaucoma; all suffered from arterio-sclerosis. The retinal vessels showed hyaline degeneration and perivasculitis. Optic-nerve excavation and obliteration of the iris-angle were not always present, but dilatation of the veins of the iris and ciliary bodies was constant. He believes glaucoma hemorrhagica to be due to an alteration of the retinal blood-vessels, secondary to a disease of the blood-vessels in general.

7. ELSCHNIG (Graz).—*Acute Retrobulbar Neuritis.*

Elschnig reports four cases of bilateral acute retrobulbar neuritis which were followed after days or weeks by acute myelitis, including encephalitis. One of the cases was subjected to post-mortem examination somewhat less than two months after the commencement of the optic neuritis, and thus the clinical diagnosis was confirmed. Acute retrobulbar neuritis begins with sudden blindness, and must be differentiated from the cases of sudden blindness with ischæmia of the retinal blood-vessels. In the former there is frequently pallor of the optic nerve and occasionally also intraocular neuritis if the retrobulbar inflammation extends to the globe. Anatomically, acute retrobulbar neuritis consists of a primary interstitial neuritis.

Uhthoff and Samelsohn took part in the discussion.

8. MICHEL (Würzburg).—*The Nerve-Fibres of the Optic Nerve and Retina.*

The axis cylinders of the nerve-fibres are connected at one point with cells and terminate in branches—terminal branches. From the axis cylinders a series of prolongations are given off—collateral branches. In the optic nerve and in the retina there are centrifugal and centripetal fibres. The terminal branches are found in the retina; they are connected with the fibres of nerve-cells, a certain amount of nourishment being given to the nerve-fibres in this manner. If one eye be enucleated in a pigeon, medullated nerve-fibres will be found on the operated side and in the optic tract, even after a considerable period; this points to centripetal and centrifugal transmission, and also to centripetal and centrifugal nourishment, the two being perfectly distinct.

Haab (Zurich) took part in the discussion.

9. SAMELSOHN (Cologne).—*Peripheral Amblyopia.*

10. WILBRAND (Hamburg).—*Disease of the Optic Nerve of Systemic Origin.*

11. E. MEYER (Paris).—*An Experimental and Clinical Contribution to Endogenous Infection of Old Prolapses of the Iris, and Protection against Ectogenous Infection.*

Meyer believes that clinical experience points to endogenous infection. In an eye which presented only corneal opacities, he observed the occurrence of herpes corneæ. In the other eye, which presented a cicatrized prolapse of the iris, opacities of the vitreous then developed. Subsequently the scar softened; hypopyon occurred and was cured by galvano-caustic treatment. This was a case of internal infection. To protect prolapses of the iris from external infection, Meyer covers them with conjunctival flaps.

12. SCHWEIGGER (Berlin).—*Correction of Myopia by Aphakia.*

Schweigger operated upon five cases—four children eight to fourteen years old, and a woman of thirty-four; all had myopia of more than 16 dioptries; he operated upon one eye, by discission, and then removed the swollen lens. Recovery was good in all cases. The optical effect of the aphakia was about 15 dioptries, but the acuteness of vision was not improved to any marked extent.

13. PFLÜGER (Bern).—*Operative Aphakia in Cases of Myopia of High Degree.*

Pflüger performed an extensive discission and removed the swollen lens substance with the lance-shaped knife. He operated in fifteen cases of myopia, varying from 12 to 18 dioptries. His results were good. Sattler (Leipzig), Landolt (Paris), and Michel are opposed to the operation; Wicherkiewicz (Posen), Thier (Aix-la-Chapelle), Samelsohn and Fick (Zurich) favor it.

14. CHIBRET (Clermond-Ferrand).—*A Study of Bilateral Paralysis of Accommodation; its Analogy to Hysterical Paralyses.*

Chibret observed thirty-four cases of bilateral paralysis of accommodation. The affection is not uncommon, and occurs most frequently in females under twenty (two thirds). In only two cases was it found in men. The range of accommodation was lessened from 2 to 4 dioptries. Acuteness of vision and refraction were almost always normal. In many cases the pupil did not respond to light. Duration of the affection was from one

to four years. Locally, convex glasses (2–3 dioptries) and cold water and electricity were used.

15. WAGENMANN (Heidelberg).—*Observations on Unilateral Retinitis Hemorrhagica.*

Wagenmann investigated a case of retinitis hemorrhagica occurring in a man of sixty, who had arterial sclerosis and who had been attacked with sudden blindness. There were numerous retinal hemorrhages; with the exception of one, all the arteries were occluded. Besides this, there was hemorrhagic infarction of the retina and multiple embolism of the arteries; there was no thrombosis of the central veins, but dilatation of the retinal veins was found.

16. HESS (Leipzig).—*The Etiology of Striped Keratitis after Cataract Extractions.*

Hess shows that the dilatation of the corneal lymph-spaces, which has hitherto been regarded as the cause of striped keratitis, is merely an accidental occurrence. The striæ result from a wave-like condition—a sort of pleating of the deepest layers of the cornea; this results from a faulty relation between the pressure in the vertical and the horizontal meridian caused by the incision in the cornea—hence a sort of compression of the cornea in the horizontal meridian. Hess was the first who succeeded in studying the striæ after experimental cataract extractions in rabbits.

17. SAMELSOHN (Cologne).—*A New Method of Operative Procedure in Symblepharon.*

18. SATTLER (Leipzig).—*Bacillus Panophthalmitis.*

Sattler found the bacillus pyocyaneus albus in a circumscribed collection of pus in the vitreous of a woman of sixty, suffering from panophthalmitis. After removal of the mycotic collections all inflammatory manifestations disappeared.

19. HAAB (Zurich).—*The Use of Strong Magnets.*

Haab places very strong magnets upon the surface of the eyeball to remove fragments of iron. In this manner these may be removed through the opening by which they entered. He always employed a large Ruhmkorff apparatus. He succeeded in three cases in which the fragments had remained in the eye for long periods and had penetrated through cornea and lens. In one case, in which the fragment had already become encapsulated in the retina, he did not succeed in removing it.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY DURING THE FIRST QUARTER OF THE YEAR 1892.

By Dr. ST. BERNHEIMER, in Vienna; Prof. HORSTMANN, in Berlin; and Dr. SILEX, in Berlin.

WITH THE CO-OPERATION OF

Dr. S. M. Burnett, Washington; Dr. Dantone, Rome; Prof. Hirschmann, Charkow; Dr. P. von Mittelstädt, Metz; Dr. Werner, Dublin; Dr. C. H. A. Westhoff, Amsterdam; Dr. Schiötz and Dr. Ole Bull, Christiania; Dr. Deus, Berlin. Dr. Herrnheiser, Prague; Dr. Sulzer, Geneva, and others.

Sections I.–XI. Translated by Dr. Ward A. Holden.

Sections I.–V. Reviewed by Dr. St. BERNHEIMER.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE.

TEXT-BOOKS, MONOGRAPHS, AND TREATISES ON GENERAL, BIBLIOGRAPHICAL, AND HISTORICAL SUBJECTS. STATISTICAL REPORTS.

1. Gosetti. The eye operations performed in the ophthalmic department of the Venice Hospital. *Annali di Ottal.*, vol. xxi., 2, 3.

2. Massachusetts Charitable Eye and Ear Infirmary. Sixty-sixth annual report, 1891.

3. Baltimore Eye, Ear, and Throat Hospital. Fourteenth annual report, 1891.

4. Oppenheimer, H. S. The blind of New York City. *Trans. Amer. Ophth. Soc.*, 1891.

5. LAQUEUR. On eye clinics and their importance in ophthalmic instruction. An address at the dedication of the university eye clinic in Strassburg, Dec. 19, 1891. Trübner.

6. COHN. Examination of the eyes of the inmates of the Breslau Institute for the Deaf and Dumb. *Centralbl. f. Augenh.* and *Zeitschr. f. Schulgesundheitspf.*, No. 3.

7. COHN. Hand-book of the Hygiene of the Eyes. Part 2. Urban and Schwarzenberg, Vienna.

8. MAGNUS. Wall charts for ophthalmic instruction. Part 1. Diagnosis of ocular disturbances of central origin.

9. HOLT. MAINE EYE AND EAR INFIRMARY. Sixth annual report, 1891.

10. BROOKLYN EYE AND EAR HOSPITAL. Twenty-third annual report, 1891.

GOSETTI (1) performed 134 cataract operations in the years 1888–91, in the ophthalmic department of the Venice Hospital, which has 112 beds. In 130 cases done with a small superior flap and with iridectomy, 119 were entirely successful, and 6 required secondary operations. In 3 cases there were complications in the interior of the eye, and in 2 cases the eye was lost by suppuration. The author emphasizes the necessity of performing iridectomy. DANTONE.

In 1891, 11,926 new patients were treated at the MASSACHUSETTS CHARITABLE EYE AND EAR INFIRMARY (2). There were 136 cataract extractions done, with suppuration in 3 cases. Graefe's operation was done in 100 cases, simple extraction in 7.

BURNETT.

At the PRESBYTERIAN EYE, EAR, AND THROAT HOSPITAL in Baltimore (3) 7,364 eye cases were seen in 1891. Of the 132 cataract extractions done, 44 were with iridectomy, and 88 without.

OPPENHEIMER (4) examined officially for the city of New York 572 persons who had reported themselves blind, 226 of whom were native-born. The principal causes of blindness were found to be as follows: Optic nerve atrophy 27, blennorrhœa neonatorum 38, blennorrhœa virulenta 93, cataract 50, choroiditis and iritis 29, glaucoma 62, trachoma 23, cataract operation 65, injuries 62, injuries of the head 22, and sympathetic ophthalmia 46.

BURNETT.

At the MAINE EYE AND EAR INFIRMARY (9) there were treated,

during 1891, 670 eye patients. A tabulated statement of the results of 29 cataract extractions with one failure is given, and a description of the new and commodious hospital building.

BURNETT.

At the BROOKLYN EYE AND EAR HOSPITAL (10) there were treated, in 1891, 6,349 eye patients. All eye operations 1,012. Extractions, simple, 2; with iridectomy, 28; in capsule, 2.

II.—GENERAL PATHOLOGY, DIAGNOSIS, AND THERAPY.

11. ANTONELLI. A contribution to the study of phthisical eyes. *Annali di Ottal.*, xx., 6, p. 471.

12. SGROSSO. A contribution to the morphology and structure of epibulbar tumors, with particular reference to the presence of psorospermiæ in and among the cells. *Annali di Ottal.*, vol. xxi., p. 3.

13. POTAPENKO. On the ætiology of the periodic eye inflammation or moon-blindness of the horse (ophthalmia periodica; irido-choroiditis recidiva; fluxion périodique of the French writers). *C. Mitwratsch*, 1892, No. 7.

14. GARNIER. On entarteritic changes in the ocular blood-vessels. *Centr. f. prakt. Augenh.*, Jan., 1892.

15. LEBER. On attenuated tuberculosis of the eye. *Ber. d. ophthal. Gesellsch.*, vol. xxi.

16. SATTLER. On bacillus ophthalmitis. *Ibid.*

17. SNELLEN, Jr. Retinal changes in naphthaline poisoning. Utrecht, 1892.

ANTONELLI (11) described in great detail the histological changes found in each tissue in a phthisical ball with beginning ossification in the shrunken and degenerated vitreous.

SGROSSO (12) gives a detailed microscopic description of sixteen epibulbar tumors removed by De Vincentiis and Morand. Three were sarcomata, one a melanoma, and twelve epitheliomata.

DANTONE.

According to SNELLEN, Jr. (17), naphthaline is absorbed from the intestinal tract, and is excreted by the kidneys. Local degenerations occur in the retina, and if its use be continued, eventually the entire retina may become affected. The degenerated retinal elements form a granular necrotic mass, which is absorbed. The pigment cells proliferate. In severe cases the pigment cells

within the focus are destroyed, and the proliferation occurs only at the margins. The process is a necrobiosis of the retina, and not an exudation between the retina and vitreous, or pigment layer (Panas); nor a collection of leucocytes in the retina (Dor); nor a hemorrhage in the chorio-capillaris (Kolinski).

The formation of cataract is a secondary process. The first stage, that of opaque spiculæ near the equator, is due to changes in the osmotic relations in and about the lens. When the use of naphthaline is stopped, and the aqueous becomes normal, and the osmotic equilibrium is restored, the lens loses its opacities. The second stage, that of opacity of the posterior cortex, is due to the fact that the nutritive fluid is not so quickly renewed on the posterior surface of the lens as on the anterior, and therefore the naphthaline contained in it remains more constant there, and acts more vigorously upon the lens. WESTHOFF.

III.—INSTRUMENTS AND REMEDIES.

18. ANTONELLI. An optometer and sciascope. *Annali di Ottal.*, vol. xxi., 2–3.

19. STRAUB. Placido's keratoscope. *Weekblad van het Nederlandsch Tijdschr. v. Geneesk.*, part 1, p. 572.

20. STRAUB. An apparatus for determining the position of rest of the eyes, and for revealing insufficiency of convergence. *Ibid.*

21. JACKSON. Cocaine anæsthesia for enucleation of the eye. *Annals of Ophth. and Otol.*, Jan., 1892.

22. STEVENS, G. T. An instrument for measuring heterophoria. *N. Y. Med. Jour.*, Jan., 1892.

23. CHISOLM. The Japanese hot-box the best means of applying dry heat for the relief of ocular inflammations. *Annals of Ophth. and Otol.*, Jan., 1892.

24. RISLEY. A new apparatus for determining and measuring anomalies of the ocular muscles. *Trans. Amer. Ophth. Soc.*, 1891.

25. RISLEY. A note on hyoscyamin. *Ibid.*

26. KNAPP. Demonstration of a roller forceps, constructed according to the principle of the mangle, for pressing out trachomatous granulations. *Ibid.*

27. AUBERT. Demonstration of a binocular corneal microscope. *Report of 21st Meeting of Germ. Ophth. Soc.*, 1891.

28. SCHWEIGGER. An electric ophthalmoscope. *Ibid.*

29. RINDFLEISCH. A simple apparatus for the objective determination of refractive errors. *Klin. Monatsbl. f. Augenh.*, vol. xxx.

30. STILLING. The use of anilin dyes. *Deutsche med. Wochenschr.*, No. 10.

31. HEDDAEUS. A trial frame. *Klin. Monatsbl. f. Augenh.*, vol. xxx.

32. LANDOLT. Clinical observations on the abuse of mercury in the treatment of diseases of the eye. *Brit. Med. Jour.*, March 26th.

33. SMITH, PRIESTLEY. On the corneal reflex of the ophthalmoscope as a test of fixation and deviation. *Ophth. Review*, vol. xi., p. 37.

The sciascopy optometer of Antonelli (18) consists of a narrow plate 20 *cm* long with fourteen small perforations containing lenses. A sliding and reversible cross-piece with additional lenses makes it possible to obtain any combination of + or − lenses between 0.5 D and 19.5 D. DANTONE.

STRAUB (19) recommends Placido's keratoscope without a magnifying glass for determining irregular and regular astigmatism above 2.5 D. WESTHOFF.

STRAUB (20) fastens a small mirror to one end of a measure 1 *m* long, and a small rule to the other end of the tape. The patient is seated with his back to the light. The observer sits opposite him and holds the mirror against his own forehead, so that the patient can look into it. The mirror is then turned on its horizontal axis and the patient fixes a distant object. The observer then makes the ordinary test with the rule. In emmetropes and myopes he found a divergent position of rest in one half the cases, and parallel axes in the other half. In hyperopes, Stilling's position of rest was either divergent or convergent, rarely parallel. He found further that hyperopes with binocular vision, as well as emmetropes, have a divergent or parallel position of rest, and that the convergent position is almost always in hyperopes not having binocular vision. With fixation for a distance of 30–50 *cm* obtained by making a black dot on the patient's nose and having him observe its reflection in the mirror,

he found that relative divergence is normal, and that marked convergence is rarely found except in hyperopes not having binocular vision. WESTHOFF.

JACKSON (21) enucleates eyes which are not hyperæmic, under the influence of cocaine. The solution is not injected into the tissues, but is simply applied on the surface as the operation proceeds. The pain is said to be but slight. BURNETT.

STEVENS (22). When a candle flame at a distance of 20 feet is observed through a 13. D lens, it appears as a round disc of light. If the lens be held before one eye while the other is left free, the image of the flame is seen in the centre of the disc in orthophoria, and out of the centre in heterophoria, its location in the latter case varying with the nature and the degree of the heterophoria. Stevens uses a lens of this strength, covered except at its optical centre, which prevents the prismatic displacement that appears when the lens is not centred. The apparatus is cheap, practical, and as accurate as any of the tests in which the apparatus is held near the eye. BURNETT.

CHISOLM (23) considers the Japanese hot-box, used for warming the hands, as a convenient means of applying dry heat to painful eyes suffering from iritis, glaucoma, and the like.

BURNETT.

RISLEY (24) exhibited a rotating prism previously described in the report of the society for 1889 ; and also a stand holding the prism, a Maddox rod, and test glasses for ametropia.

RISLEY (25) concludes from his studies in regard to the chemical constitution of hyoscyamin salts, that only the pure crystals should be used, that the solution prescribed should be neutral and prepared with only moderate heat, and finally that alkaline filter paper should not be employed. BURNETT.

In constructing forceps for the expression of trachoma granules, KNAPP (26) has followed the principle of the mangle, by which method the contents of the granules are forced out with a minimum amount of tearing of the tissues. He has used the instrument in a great number of cases with favorable results. This variety of forceps is particularly serviceable in cases of thick spawn-like granulations, which are in most instances cured at a single sitting. BURNETT.

STILLING (30) reviews the numerous papers written in the last two years, both favorable and unfavorable, regarding the employment of anilin, and holds to his original belief that pyoctanin,

rightly used, cuts short suppuration. The objection of Liebreich that the agent is not a purely chemical one, has not been verified. The results of its application depend principally upon the manner of using it. The much better results that are observed in severe corneal affections with ulceration, and the poorer results got in corneal inflammations where the affected part is covered with a healthy layer, prove that the remedy is of value when it can have free access to the purulent parts, and an intense staining can take place.

LANDOLT (32) is convinced of the inefficacy of mercurial treatment in tabic optic-nerve atrophy, and in cases of choroiditis and chorio-retinitis with marked atrophic changes. WERNER.

PRIESTLEY SMITH (33) shows how the deviation of an eye may be determined by observing the corneal reflex of the ophthalmoscope in its relation to the pupil. This method is sufficient for judging the immediate effect of a tenotomy. WERNER.

IV.—ANATOMY.

34. NUEL. On the vascular supply of the choroid and the nutrition of the retina, particularly in the region of the fovea centralis. *Arch. d'Ophth.*, vol. xii., Feb.

NUEL (34) describes the vascular system of the choroid in its different segments, and considers Sattler's description too schematic. The best eyes for such studies are those which have been enucleated for foreign bodies in the anterior segment, before a plastic exudation has taken place. In such eyes the vessels are well filled and the interstitial œdema allows the ready separation of the individual layers. The following points may be mentioned particularly. Toward the ora serrata the larger vessels are fewer in number; toward the macula, on the contrary, they form an unbroken layer. In the layer of larger vessels the arteries predominate; in the layer of smaller vessels, the veins. The intervascular tissue is thicker near the inner surface of the choroid, and particularly so in the neighborhood of the macula. Small veins and capillaries are present near the macula in such number that the choroid is thicker here than in any other part. The arrangement of the vessels provides for the nutrition of the outer layers of the retina, and the fovea is nourished exclusively from this source. The choroidal vessels thus serve the photochemical function of the retina, while the retinal vessels furnish its nutrition in

a general sense. In pathological conditions of the macula, the vascular distribution plays an important rôle.

V.—PHYSIOLOGY.

35. SNELL. On the importance of the examination of the eyes separately for defects of color-vision. *Brit. Med. Journ.*, Jan., 1892.

36. GULLSTRAND. On the simultaneous determination of refraction and visual acuity. *Hygiea-förhandl*, p. 73; *Nord. med. Arch.*, vol. xxiii., Stockholm.

37. SULZER. The form of the human cornea and its influence on vision. Part 2. Influence of the cornea on vision. *Arch. d'Opht.*, vol. xii., No. 1.

38. MAKLAKOFF. Contributions to the study of the influence of voltaic light on the skin. *Arch. d'Oph.*, vol. xii., No. 3.

39. TSCHERNING. Note on a hitherto unrecognized change which the lens undergoes in the act of accommodation. *Arch. d'Oph.*, vol. xii., No. 3.

40. SCIMEMI. On the conduction of the tears. *Annali di Ottal.*, vol. xxi., No. 2–3, p. 222.

41. OSIO. The circulation of the intraocular fluids; nutrition of the vitreous. Critical examination of the recent studies on the subject. *Annali di Ottal.*, vol. xxi., No. 2–3, p. 168.

42. MATTHIESSEN. The recent progress in our knowledge of the optical structure of the vertebrate eye. *Von Helmholtz Festschrift.* Leipsic, 1891.

43. UHTHOFF. Study of the visual education of a child born blind, and successfully operated at the age of seven. *Ibidem.*

44. KNIES. Contribution to the study of ocular estimation of size. *Ibidem.*

45. ENGELMANN. On electrical processes in the eye with reflex and direct irritation of the optic nerve. *Ibidem.*

46. KÖNIG. On the values of spectral colors with different degrees of absolute intensity. *Ibidem.*

47. FICK. On fatigue and restoration of the retina. A reply. *Graefe's Archiv*, vol. xxxviii., 1.

48. HEBOLD. Decussation of the optic-nerve fibres in man. *Ibidem.*

49. SCHNELLER. On the motions serving binocular vision. *Ibidem.*

50. DIMMER. On the reflex from the retinal vessels. *Report of 21st Meeting German Ophth. Soc.*, 1891.

51. DIMMER. The ophthalmoscopic retinal reflexes. *Ibidem.*

52. SACHS. The influence of colored lights on the size of the pupil. *Arch. f. d. gesammte Physiologie*, vol. lii.

53. SACHS, H. On optic after-images. *Centr. f. Nervenheilkunde*, vol. xv., 2.

SNELL (35) reports a case of unilateral green blindness without a defect in the visual field, and a second case with red blindness in one eye and incomplete green blindness in the other.

WERNER.

GULLSTRAND (36) shows that we may obtain much simpler relations for the visual acuity in different refractive conditions and in accommodation, by using either the angle whose vertex lies at the anterior principal point—the principal angle, or the angle whose vertex lies at the anterior focal point—the focal angle, in place of the visual angle usually employed, whose vertex lies at the anterior nodal point. SCHIÖTZ.

As was shown in the first part of SULZER'S (37) paper, the peripheric decrease in corneal curvature is not the same for equal distances from the point where the line of vision intersects the cornea. From which fact it follows: (1) that there being no astigmatism in the centre, the peripheric portions of the cornea are astigmatic; (2) that with central astigmatism of low degree, there is no peripheric astigmatism or there is astigmatism against the rule; (3) that with central astigmatism against the rule, the peripheric astigmatism is greater and also against the rule; (4) that with moderate or excessive central astigmatism there is a more excessive peripheric astigmatism. This relation holds for all cases excepting those in which there is no central astigmatism. As in the ordinary method of examination we do not compare the astigmatism found by central fixation with that of the corneal zone concerned in direct vision, we may explain the discrepancy between the subjective and the objective astigmatism by this dissymmetry of the cornea, without having recourse to a lenticular astigmatism, which, until demonstrated by direct measurement, Sulzer will not accept. The location of the pupil, whose centre lies on an average 5° to the temporal side of the point

where the line of vision intersects the cornea, and still more the artificial dilatation of the pupil, have a marked influence on the degree of subjective astigmatism. An ordinary cylindrical glass can only correct the astigmatism of a particular segment of the cornea, and will be of the greater benefit the less dissymmetry there is of the cornea. v. MITTLESTÄDT.

MAKLAKOFF (38) studied the action of the electric light on the eye and the skin, and devoted particular attention to the agents which prevent the injurious effect of the actinic rays. The best of these agents are quinia solutions and red or yellow varnish. When the skin was pencilled with one of these, the light had no effect on it, but pencilling with green or blue varnish or with glycerin, in similar conditions, did not prevent, but rather favored the production of the well-known signs of insolation. In Maklakoff's opinion it is the terminations of the vasomotor nerves and the walls of the capillaries, which, in a manner not yet understood, are affected by the actinic rays, and produce the changes which are so sharply localized in the regions exposed to the light. v. MITTELSTÄDT.

TSCHERNING (39) studied the changes of form in the lens in the act of accommodation by means of his ophthalmo-phakometer, and found that the Purkinje images are displaced upward, a phenomenon which can only be explained by supposing that toward the end of the act of accommodation the lens is displaced downward, thus producing a more accurate centring of the optical apparatus. v. MITTELSTÄDT.

SCIMEMI (40) made a number of experiments with syphons and capillary tubes, and also tried in cases of lachrymal fistula and normally functionating lachrymal apparatus to suck the tears through the lachrymal canaliculi. He concludes that the lachrymal sac is always at least half filled with fluid. The tears pass into the sac by capillary attraction and by aspiration of the sac itself, which is dilated before and after the closure of the lids by the lachrymal muscles. This dilatation stands in direct relation to the muscular contraction. It is weakest in the simple closure of the lids, more marked in firm closure of the lids, and reaches its maximum when the lids are pressed together. The tears flow down into the nose from the action of gravity, aided by the elasticity of the walls of the sac. DANTONE.

OSIO (41) speaks in detail of the gradual development of our knowledge concerning the circulation of the intraocular fluids,

their source, their course in the interior of the eye, and their exits; he dwells on the influence exerted on their circulation by the blood, the nervous system, the accommodation, by pathological changes in the eye, paracentesis of the cornea and iridectomy, and finally by the local application of remedies. The experiments and opinions of many authors are collected and compared. In conclusion, the author collates the present views on the nutrition of the vitreous. DANTONE.

MATTHIESSEN (42) has with great care collected the scattered facts which are known in regard to the optical structure and the fundamental principles of the dioptrics of the vertebrate eye, and chiefly those concerning the refractive index of the ocular media, and the curvature and position of the refracting surfaces. For the most part only a few individuals of a species have been examined, so that the result cannot be considered to be unconditionally correct for all.

Each medium is considered in turn, and in general tables the values for the different species are compared. The metric and theoretic results of these comparisons are applied to the dioptrics of the enlarged eye. (BLAUWAL).

In UHTHOFF'S (43) blind-born, seven-year-old patient, an iridectomy upward was made in each eye, followed by an iridectomy downward. The shrunken cataract was removed from the right eye, but in the left a sufficiently large clear space was obtained by the second iridectomy. Observations of the patient were made for three months. Before and immediately after the operation the boy was quiet and apathetic, a change in his entire psychical condition being only observed much later.

KNIES (44) reports a series of interesting experiments designed for the purpose of studying the peculiar relations in the matter of sense memory and ocular estimation of size.

ENGELMANN (45) by new experiments sought to confirm the theory that there are both centripetally and centrifugally conducting optic-nerve fibres. Experiments were made first on the reflex irritation of the optic nerve, both by light and by chemical agents, on curarized frogs. To avoid possible deviations of the current, experiments were also made on the bare skull. The results of the experiments were positive. To prove that there had been no indirect irritation, the experiments were repeated after cutting the optic nerve. In the latter instances the result was negative or doubtful.

In a second series of experiments, light was avoided and chemical irritation with crystals of sod. chlor. was found to produce a positive result. When the chiasm was cut off from the brain there was no result.

The experiments on direct stimulation of the optic nerve were also made with both light and chemical agents. The centre of the cornea was employed, $\frac{3}{4}$ *mm* from the nerve entrance. The results were always positive. In order to exclude simultaneous irritation of the retina, the cut end of the optic nerve was also irritated with salt crystals, with positive effect.

KÖNIG (46) undertook to measure and compare the values (luminous intensities) of the spectral colors with different degrees of actual intensity. With differences of intensity both the curves of the aperature breadth and the curves of the values changed their form.

Sections VI.–XI. Reviewed by PROF. HORSTMANN.

VI.—ERRORS OF REFRACTION AND ACCOMMODATION.

54. BATTEN, R. D. Myopia the result of constitutional disease. *Ophth. Rev.*, vol. xi., 1, 1892.

55. FUKALA. On the cause of the increase in visual acuity in aphakic eyes which have been highly myopic. *Berlin. klin. Wochenschr.*, No. 23, 1892.

56. FUKALA. The injurious influence of the accommodation on myopia. *Berl. Klin. Wochenschr.*, No. 23, 1892.

57. LANDSBERG. A peculiar employment of convex glasses in excessive hyperopia. *Klin. Monatsbl f. Augenh.*, xxx., p. 187.

58. HARLAN. A further note on the use of toric lenses in astigmatism. *Trans. Amer. Ophth. Soc.*, 1891.

59. WALTON, G. L. and CARTER, C. F. Over-exertion of the eyes and headache. *Med. News*, March 19, 1892.

60. THEOBALD. Subnormal accommodative power in young persons a not unusual cause of asthenopia. *Trans. Amer. Ophth. Soc.*, 1891.

61. DE SCHWEINITZ. Remarks on asthenopia and intra-nasal disease. *Med. News*, Apr. 2, 1892.

62. GOULD. Severe "common cold," due to reflexes from the eye. *Med. News*, March 12, 1892.

63. Roosa. Astigmatism, its relative importance in asthenopia due to errors of refraction. *Med. Rec.*, March 26, 1892.

64. Cheney, F. E. Ocular headaches. *Boston Med. and Surg. Jour.*, Jan. 7, 1892.

65. Derby, Hasket. Macular changes as influencing the prognosis in advancing myopia. *Trans. Amer. Ophth. Soc.*, 1891.

Batten (54) is of the opinion that a high tension of the pulse and other vascular changes are common causes of myopia. Eleven cases are cited in support of this view, at the head of which stands myopia and chronic bronchitis in a child (!). Werner.

Fukala (55), who performed discission in a number of cases of high myopia, found an increase in visual acuity after the operation. The cause of this condition is referred to the fact that the second nodal point comes to lie farther from the retina, and therefore objects appear larger and nearer. He finds that the refraction of the myopic eye is decreased, on an average, 15.5 D in consequence of the aphakia. From this he concluded that in myopia of high degree, not only the increase in axial diameter of the eye, but also an increased refractive index of the lens, plays a rôle in the production of the myopia.

Fukala (56) regards the doing away with accommodation in excessive myopia as an advantage, since myopia is increased by the raised tension which accommodation produces.

Landsberg (57) reported the case of an individual with congenital aphakia, who has $V = \frac{5}{30}$ with a $+ 8$. D glass when it was held 6 *cm* from the eye. If the glass was held near the eye, vision was not improved by it.

Harlan (58) reported a case in which he prescribed toric lenses, $- 5.5$ D $- 2.5$ D_c 65°, and $- 4.5$ D $- 4$. D_c 130°. The glasses made by the optician were not toric, but had the ordinary form and were so uncomfortable that they could not be worn. They were exchanged for toric lenses, which were worn with comfort. Burnett.

Walton and Carter (59) conclude from the study of nervous patients that a large percentage of cases of functional headache and particularly migraine are due to eye strain, and further that such symptoms occur even with normal visual acuity, so that when possible even the slightest error of refraction in such patients should be corrected. Burnett.

THEOBALD (60) believes that when there is exophoria of more than 3° with fixation for 13″, and orthophoria for distance, there is a weakness of accommodation. The weakest convex lens that gives the normal exophoria of 3° for near, should be prescribed for near work. BURNETT.

DE SCHWEINITZ (61) reports three cases of asthenopia with ametropia and heterophoria which were not improved by the correction of these errors, but were benefited by treatment of a nasal catarrh. BURNETT.

GOULD (62) gives a case of a strong man of forty-six, who contracts a severe cold whenever he goes without the glasses which correct his astigmatism and a part of his myopia, and the same thing occurred when he wore the glasses giving complete correction to his myopia, and when the cylinders were improperly set. BURNETT.

ROOSA (63) gives the histories of eleven cases to support his theme that asthenopia is due mainly not to hypermetropia or myopia or muscular insufficiency but to astigmatism; and insists on the almost complete uselessness of paralyzing the accommodation when the ophthalmometer can be used. His experience would seem to be unique in these particulars. BURNETT.

CHENEY (64) relates three cases to support his theme that patients may suffer from ocular headache for years and have little if any local eye symptoms. The sight may be good and they may be able to use their eyes constantly for near work with little discomfort referable to the eyes and yet suffer from migraine which has its origin in a refractive trouble. BURNETT.

DERBY (65) has found that those cases of progressive myopia in which there was a pigment patch in the macular region never went on to separation of the retina even though the myopia and other changes in the interior of the ball were excessive. He relates a typical case in point of a patient who had been under observation for thirty years. He attributes the protection against detachment to a probable adhesion between the choroid and retina at that point. BURNETT.

VII.—LIDS.

66. STRAUB. Ophthalmia scrofulosa. *Weekblad Nederlandsch Tijdschr. v. Geneeskunde*, 1892, 1, p. 664.

67. GURFINKEL. Malignant pustule of the eyelids. *Wjestnik Ophth.*, ix., 1., p. 23.

68. HIRSCHBERG. Vaccine ulcer of the lid. *Centralbl. f. prakt. Augenheilk.*, 1892, p. 17.

69. WEYMANN. Fungus chalazicus. *Ophth. Rec.*, i., 9, p. 307.

70. HIRSCHBERG. On the formation of eyelashes. *Centralb. f. prakt. Augenheilk.*, 1892, p. 131.

71. STOEWER. Cysts of the upper lids. *Zehender's klin. Monatsbl.*, vol. xxx., p. 192.

72. HOHENBERGER. Pigmentary nævus of the lid with beginning sarcomatous degeneration. *Graefe's Archiv.*, vol. xxxviii., 2, p. 146.

73. DE SCHWEINITZ. Neuroma of the right upper lid and the neighboring temporal region. *Trans. Amer. Ophth. Soc.*, 1891.

74. VOSSIUS. Two cases of congenital, almost total immobility of the eyes and the upper lids. *Beiträge zur Augenheilk.*, 1892, part 5.

75. BIRNBACHER. A new operation for congenital ptosis. *Centralb. f. prakt. Augenheilk.*, 1892, p. 131.

76. GUIBERT. A case of double congenital entropium; cure. *Arch. f. Opht.*, vol. xii., No. 2, p. 101.

77. FRANKE, E. Remarks on skin and mucous membrane transplantation in trichiasis operations. *Centralbl. f. prakt. Augenheilk.*, 1892, p. 34.

78. SCELLINGO. On plastic operations on the lids. *Boll. d. R. Accad. Med. di Roma*, vol. xviii., No. 1.

79. GIFFORD. On the use of Thiersch flaps in the treatment of trichiasis and entropium. *Amer. Journ. of Ophth.*, 1892, No. 1, p. 1.

80. THEOBALD. Two successful cases of skin transplantation by Thiersch's method. *Trans. Amer. Ophth. Soc.*, 1891.

81. ZINN. On reversal of the margin of the lid containing the eyelashes in trichiasis. *Wiener klin. Wochenschr.*, 1892, No. 21.

82. HARLAN. A modification of Dieffenbach's operation for restoration of the lower lid. *Trans. Amer. Ophth. Soc.*, 1891.

83. HANSELL. Pagenstecher's operation for entropium and distichiasis. *Med. News*, Feb. 13, 1892.

STRAUB (66) found the staphylococcus pyogenes aureus constantly in cultures from the fluid beneath blepharitis crusts and eczematous crusts on the ears and nose. WESTHOFF.

In GURFINKEL'S (67) severe case of malignant pustule of the lid, the successful treatment consisted principally in local subcutaneous injections of two per cent. carbolic solution. The defect remaining in the lid was not large. HIRSCHMANN.

HIRSCHBERG (68) in a woman of thirty-eight observed a vaccine ulcer on the upper lid. She had been infected with the lymph of her one-year-old infant who had been vaccinated. The ulcer healed in twelve days.

WEYMANN (69) believes that a chalazion is a true granuloma of parasitic origin. He pictures and describes a fungus which he has found in the contents of the cyst. BURNETT.

In order to obtain lashes in plastic operations of the upper lid, HIRSCHBERG (70) carries the lower margin of the forehead flap lengthwise through the eyebrow, so that when the lower margin of the flap comes to form the free margin of the lid the hairs of the brow play the rôle of lashes.

STOEWER (71) gave an anatomical description of two cysts which were removed from the upper lids of a girl of sixteen. He believes them to have arisen from the glands of Krause.

HOHENBERGER (72) examined a pigment nævus with beginning sarcomatous degeneration, which extended over the upper and the lower lid of a girl of sixteen. Three years after the removal there was no sign of recurrence.

DE SCHWEINITZ'S (73) case was that of a boy of twenty, who had a congenital tumor of the right eyelid causing pronounced ptosis, which extended also to the temporal region, giving rise to clinical appearances similar to the condition known as elephantiasis. Examination of the specimen, however, revealed, in addition to a general hypertrophy of the glandular tissue of the skin, some large masses which on cross-section appear as whorls of soft fibrous tissue containing in their centre more or less degenerated nerve-fibres. Excellent micro-photographs of the sections are given, as well as drawings showing the condition of the patient before and after the operation, which was, in result, fairly successful. BURNETT.

VOSSIUS (74) observed an almost total immobility of both eyes and of the upper lids in two brothers of healthy family. The visual acuity was considerably reduced in both. Vossius attributes the affection to a muscular anomaly.

In operating for ptosis, BIRNBACHER (75) makes an incision in the skin of the lid along the entire upper margin of the tarsus.

Three double sutures are passed through the margin of the tarsus and are then carried under the skin and are brought out in the brow and knotted. The loops of thread are left for 20–25 days, producing subcutaneous cicatrices between the brow and the tarsus. The lid may then be raised by the frontalis muscle.

GUIBERT (76) saw in a girl of three a congenital entropium of the lower lids, the margins of which were apparently thickened and without lashes. When the lids were everted the lashes were brought into view. The eyes showed no signs of irritation and the corneæ were clear. The lids were burned through to the conjunctiva in a line 2 *mm* from the lid margin and parallel to it, and on the following day the position of the lids was normal. The tarsal cartilage was wanting. The child's skull was asymmetrical. The mother was a tippler and phthisical; the father, sound.

V. MITTELSTÄDT.

For trichiasis, FRANKE (77) transplants skin flaps from the upper arm into the split intermarginal space. If mucous membrane is used, it is taken from the lower lip.

SCELLINGO (78) reports two successful plastic operations for cicatricial ectropium. Skin flaps were taken from the inner surface of the upper arm to cover the defect. DANTONE.

After splitting the lid as in the ordinary Jaesche-Arlt operation, GIFFORD (79) makes an incision in the skin containing the hairs at an angle of 45° at each end of the cut, extending $\frac{1}{2}$ *cm.* This allows of a considerable retraction of the detached skin, which is held in place by sutures, and the raw surfaces thus exposed he fills with grafts from the inner side of the forearm after Thiersch's method. He prefers these grafts to those from the lips, and in 28 cases none have failed in which it was given a fair trial. He shaves the graft from the skin with a broad razor. He has also modified v. Burow's operation of the undercutting of the tarsus, by filling in the opening with grafts from the lips.

BURNETT.

THEOBALD (80) has used skin-grafting after Thiersch's method in two cases which, after the lapse of months, seem to be successes. The grafts, which were taken from the inside of the forearm, all "took" except one, and that sloughed only at one part. Both were treated as out-patients and with no unusual antiseptic precautions. BURNETT.

The reversing of the tissue containing the eyelashes, which operation Stellwag performs for distichiasis and trichiasis, had

the disadvantage that the lashes in time fell out of the atrophic follicles. ZINN (81) guards against this by cutting out a broader piece of skin, which, before the transplantation, is freed from all bits of tarsus and of a portion of the muscle, without injuring the hair follicles.

HARLAN (82) fills in the large exposed surfaces left in the malar surface by the sliding inward of the flap in Dieffenbach's operation, by a flap from the temporal region, which he slides downward. The result, he claims, is most satisfactory, the resulting deformity being rarely noticeable. BURNETT.

HANSELL (83) reports two successful operations for entropium by Pagenstecher's modification of Hotz's operation.

BURNETT.

VIII.—LACHRYMAL APPARATUS.

84. HEDDAEUS. On the so-called blennorrhœa of the lachrymal sac in the new-born. *Zehender's klin. Monatsbl.*, xxx., p. 81.

85. WEISS, L. Treatment of the lachrymal passages of the new-born. *Ibidem*, p. 238.

86. MERCANTI. A contribution to the etiology of dacryocystitis in the new-born. *Annali di Ottal.*, xxi., 2–3, p. 133.

87. GUAITA. A new operative procedure for the rapid treatment of dacryo-cystitis. *Annal. d'Ocul.*, cvii., p. 30; *Centralbl. f. prakt. Augenh.*, 1892, p. 5.

88. GIULINI. A case of small round-celled sarcoma of the lachrymal gland in each eye. *Munchener med. Wochensch.*, 1892, No. 6.

89. GOULD. A simple method of treating many cases of lachrymal obstruction. *N. Y. Med. Jour.*, June 4, 1892.

According to HEDDAEUS (84) the blennorrhœa of the lachrymal sac in infants is nothing but a stagnation of the physiological secretion of the tear passages caused by the mechanical stoppage of the nasal orifice of the lachrymal duct.

In the experience of WEISS, (85) simple digital expression of the contents of the lachrymal sac is often not sufficient in the new-born, and the repeated passage of conical sounds is indicated.

MERCANTI (86) in a two-day-old infant observed a tumor of the lachrymal sac, while no pathological change could be discovered in the eye or in the nasal passages. Cultures of the thick pale

yellow pus showed a peculiar bacillus which after various culture and inoculation experiments, the author took to be a variety of the bacterium coli commune. The author assumed an infection at birth from the fæces of the mother. The infant recovered quickly with antiseptic treatment and continuous expression of the sac, and particularly after the twelfth day when the lachrymal bone seemed to be perforated and the pus discharged into the nasal cavity. DANTONE.

For the purpose of healing a dacryocystitis quickly, GUAITA (87) advises scraping the cavity of the lachrymal sac and introducing a canula of decalcified bone into the lachrymal duct which has been previously split.

GIULINI (88) in a man æt. seventy-seven, observed a tumor of the lachrymal gland in each eye. Both glands were extirpated and the tumor was found to be a small-celled sarcoma.

GOULD (89) recommends, in the treatment of many cases of dacryocystitis and lachrymal obstruction, frequent emptying of the sac, with massage, in preference to opening the canaliculus. Only in more obstinate cases does he advise the dividing of the sphincter of the punctum. BURNETT.

IX.—MUSCLES AND NERVES.

90. GRUENING. On the operative treatment of divergent squint. *N. Y. Med. Jour.*, March 12, 1892.

91. STEVENS. An examination of some of the means for the determination of heterophoria. *Ophthalmic Record*, Jan.-Feb., 1892.

92. STEVENS. Sudden attacks of diplopia. *Annals of Ophth. and Otol.*, Jan., 1892.

93. SAVAGE. Heterophoria, a safe line drawn between operative and non-operative cases. *Ophth. Rec.*, Jan.-Feb., 1892.

94. BURNETT, SWAN M. Contributions to the study of heterophoria and its relations to asthenopia, headache, and other nervous symptoms. *Trans. Amer. Ophth. Soc.*, 1891.

95. BOEDEKER. On a case of chronic progressive paralysis of the ocular muscles, in conjunction with toxic amblyopia. *Arch. f. Psych.*, xxiii., 2, p. 313.

96. FRANK. On ophthalmoplegia interior. *Vereinsblatt d. Pfälzer Aerzte*, 1892, p. 2.

97. WIDMARK. On ocular symptoms with trigeminus affections. *Nord. Ophthalm. Tidsk.*, ii., p. 63, Copenhagen, 1891.

98. HOWE. Concerning muscular advancement. *Amer. Jour. of Ophth.*, May, 1892.

In absolute divergent squint not periodic, paretic, or secondary, GRUENING (90) exposes the tendon of the externus in its whole extent and when the amount of the divergence is not more than 2 *mm*, simply divides the tendon at its attachment. When it exceeds that amount he divides it farther back from its attachment, in proportion to the degree of squint. If, for example, there is 5 *mm* divergence, he divides the tendon 5 *mm* from its attachment. He operates on both externi at the same time, drawing the eyes inward by means of sutures which are passed through the conjunctiva above the interni and are knotted over the nose. BURNETT.

STEVENS (91) thinks that we cannot have too many methods for testing the balance of the ocular muscles and that all the proposed methods have a special value in certain cases. The deviation in exclusion including the parallax, while in some cases an invaluable test, is not a ready one and one not always available. The Maddox rod is useful and very convenient. The lens with a sptenopaïc opening which causes the image of the eye before which it is held to appear as a disc of diffused light, seems the best of all those which are used near the eye. The diplopia by a simple prism, especially if held close to the eye, is unsatisfactory. However, the phorometer is the test on which he relies principally for information, the other tests being auxiliary. BURNETT.

STEVENS (92) relates the histories of two cases which serve as the basis of some observations on sudden attacks of diplopia, and concludes that the occurrence of diplopia, without paralysis, in persons accustomed to single vision does not necessarily call for operative interference. In such cases the general condition of the patient must be gone over carefully, and the function of the two eyes as regards their muscular power tested separately and together. BURNETT.

In heterophoria, SAVAGE (93) considers that primarily all cases which show diplopia under the red-glass test are subjects for operation, and in a general way hyperphoria of 3° and exophoria or esophoria of 10° or more are also operative cases, and finally all cases even of low degrees, which are not benefited with prisms, should be operated on. BURNETT.

BURNETT (94) reports six cases in which he had made tenotomies for heterophoria accompanied with severe asthenopia and headaches, one of hyperphoria, one of esophoria and four of exophoria. In all, the symptoms disappeared. All had been treated previously with prisms. BURNETT.

WIDMARK (97) discusses a series of cases having ocular symptoms with affections of the trigeminus. They showed pain in one or both eyes, with or without simultaneous headache. Often there was photophobia, lachrymation, and pain in the lids. The symptoms appeared most frequently with near work and artificial illumination. There is a feeling of oppression in the forehead and sensitiveness along the nerves. The treatment consists in massage of the sensitive points. SCHIÖTZ.

HOWE (98) has modified Wecker's forceps for holding the divided tendon in advancement, by making it much smaller and lighter so that it can be left to itself when it has once clasped the tendon. He uses the loop suture of Prince with some slight modifications. BURNETT.

X.—ORBIT AND NEIGHBORING CAVITIES.

99. HODGES, F. A case of tenonitis after influenza ; excision. *Ophth. Rev.*, vol. xi., No. 124, p. 149.

100. USZYNSKI, A. On retrobulbar affections. *Zehender's klin. Monatsbl.*, vol. xxx., p. 110.

101. BORTHEN, L. Localized orbital abscess as a complication of influenza. *Norsk. Mag. f. Lagerid.*, 1891, No. 3, p. 203.

102. MILLIKEN. A case of partially bony tumor of the orbit ; removal ; recovery. *Trans. Amer. Ophth. Soc.*, 1891.

103. GAYET. On the osseous restoration of the margin of the orbit. *Arch. d'Opht.*, vol. xii., 4, p. 93.

In HODGES' (99) patient, chemosis and proptosis of the right eye appeared fourteen days after an attack of influenza. Incision had no effect. The eye was quite blind. The excision was difficult on account of the adhesions. These consisted of fibrous masses with numerous round cells, which surrounded the inferior portion of the eye. The ciliary body was also infiltrated with round cells. Besides this there was total detachment of the retina. WERNER.

USZYNSKI (100) reports a case of retrobulbar abscess which got well after incision.

BORTHEN (101) reports the case of a girl of fourteen in whom, after an attack of influenza, the right eyelids became œdematous, the ball prominent and immobile, and an abscess formed in the nasal portion of the orbit. Recovery after incision. Vision remained normal. SCHIÖTZ.

In MILLIKEN'S (102) case there was a hard tumor at the upper inner portion of the orbit pushing the ball outward, downward, and forward. This was first noticed at six years of age as a small tumor, which was operated on, but there had been a discharge ever since. On exposing it by an incision in the integument it was found to be hollow and contained a soft, fibrous material well organized, but no pus. The walls were cut away, the contents removed, and the cavity thoroughly cleansed and made aseptic. The external wound was closed after the orbital contents had been put in position. The wound healed nicely and there only remains some ptosis. Movements of the eye are limited ; there is diplopia and the nerve is whitish. The contents of the tumor were found to be sarcomatous. BURNETT.

GAYET'S (103) case was that of a young man who had been struck on the lower margin of the orbit with a piece of wood. There was ectropium of the lower lid produced by adherence of the lid to the depressed bone cicatrix at the inferior orbital margin. Gayet cut round the cutaneous cicatrix, loosened the skin of the lid with the periosteum and pushed it upward. The orbital margin, which now lay bare, was separated from the superior maxilla by boring a number of holes in a line convex upward with a drill, and filing through the remaining communications. The bony plate, which now was adherent only to the floor of the orbit, was pushed upward and held in its new position by three platinum clamps fastened into the superior maxilla, thus leaving a free opening into the maxillary antrum. The lid flap was then brought down and fastened. Recovery without fever or accident. Result very satisfactory. V. MITTELSTÄDT.

XI.—CONJUNCTIVA, CORNEA, SCLERA, ANTERIOR CHAMBER.

104. BRISKEN. The prophylaxis of ophthalmia neonatorum. *Münch. med. Wochensch.*, No. 5, 1892.

105. V. STEINBÜCHEL, R. The influence of gonorrhœa on the puerperal woman, and on the eye diseases of the new-born. *Wiener klin. Wochensch.*, Nos. 21, 22, 1892.

106. WILSON, F. M. On the use of vaseline in gonorrhœal conjunctivitis. *Trans. Amer. Ophth. Soc.*, 1891.

107. PANAS. On the treatment of trachoma; with an historical sketch. *Arch. d'Opht.*, vol. xii., No. 6, p. 358.

108. GERKEN. The trachoma question. *Wjest. Oph.*, vol. ix., No. 3, p. 197.

109. DARIER. Surgical treatment of trachoma. *Arch. d'Opht.*, vol. xii., No. 2, p. 95.

110. GERKE, O. A case of croup of the conjunctiva, mouth, and nose. *Arch. f. Augenheilk.*, vol. xxiv., p. 305.

111. KAIN, E. On the etiology of croupous conjunctivitis. *Wiener med. Wochenschr.*, No. 10, 1892.

112. BARNES, S. D. Mucous patch of the conjunctiva. *Amer. Journ. of Ophth.*, Feb., 1892.

113. WEYMANN, M. J. A case of fibro-lipoma of the conjunctiva. *Ophth. Record*, No. 1, 1892.

114. SHIRLEY, I. A. Fatal hemorrhage in an infant after scarification of the conjunctiva. *N. Y. Med. Journ.*, Jan. 2, 1892.

115. FUCHS. On pterygium. *Græfe's Archiv.*, xxxviii., No. 2, p. 1.

116. CLAIBORNE, J. H. The treatment of trachoma. *Ann. Oph. and Otol.*, Jan., 1892.

117. WOLFNER, H. T. How to treat simple conjunctivitis and purulent ophthalmia. *Ann. Oph. and Otol.*, Jan., 1892.

In women who are seen at the beginning of labor, BRISKEN (104) advises the use of disinfecting washes in the vagina according to Kaltenbach's method, and the washing out the eyes of the newborn infant with distilled water; in women who first come under observation when labor is far advanced, Credé's method must also be employed. There is no means of protection against late infection.

V. STEINBÜCHEL (105) examined the genital secretion of 338 pregnant women bacteriologically, and found positive evidences of gonorrhœa in 73. Six of the infants born of the 73 infected women suffered from ocular affections, of which 3 cases were virulent and 3 not. Of the total 313 infants born, 4 had virulent and 9 benign catarrhs. In the 4 cases of virulent catarrh, the gonococcus was found, yet only 3 cases were due to infection at birth. The greater part of the ocular affections were irritation

catarrhs and slight inflammations, which were caused by the careless employment of Credé's method.

Wilson (106) has used introduction of vaseline under the lids as an adjuvant in treating purulent conjunctivitis, and with markedly good results. He considers it efficient by its cleansing powers primarily, though it possibly may have a curative one as well. Burnett.

Panas (107), after a short historical review of the treatment of granulations, gives his own treatment without advancing anything particularly new. v. Mittelstädt.

Gerken (108) in 1890 examined the eyes of all the inhabitants of the village of Lichterma. He considers the cause of trachoma to be the immediate inoculation by the transfer of the fresh secretion of trachomatous eyes from one subject to another. The trachoma begins with the formation of follicles. In a fourth of the cases the trachoma gets well of itself, leaving small cicatrices at the orbital margin of the tarsus, which do not injure the eye. Hirschmann.

Darier (109), after an experience of 130 cases in Abadie's clinic, recommends the surgical treatment of granular conjunctivitis. Under ether the lid is everted with a particular forceps, the conjunctiva scarified, then with a sharp spoon and small brush all morbid tissue is removed, and finally the entire surface is thoroughly washed with a 1:500 sublimate solution. After this ice compresses and bathing with 1:2000 sublimate solution. The lids must be everted daily, the conjunctiva cleaned with a sublimate solution, any adhesions loosened, and when there are still granules, the brushing must be repeated. The treatment lasts a month on the average. If the lids are not everted and cleaned daily, symblepharon may occur. In 10 cases an entropium had to be corrected. The cicatrices are not more marked than those following other methods of treatment. In many cases there is a fibrinous exudation on the conjunctiva and rarely corneal affections. If such have existed previously, they are favorably influenced by this treatment. v. Mittelstädt.

Gerke (110) observed in a soldier of twenty-one the appearance of typical croup of the conjunctiva, which later affected the mucous membrane of the mouth and nose. Inoculations of bits of the membrane in agar-agar, blood serum, and gelatine, produced cultures of double bacilli which stained best with methylin

blue. Inoculations of these bacilli on the conjunctiva of rabbits and men produced a non-membranous conjunctivitis.

KAIN (111) had a similar result.

In a man with a history of syphilis, under BARNES' (112) care, a mucous patch was seen on the conjunctiva of the lower lid of the left eye, which extended in a few days to the conjunctiva of the globe. It disappeared under the use of black wash and proper internal treatment. BURNETT.

The tumor described and removed by WEYMANN (113) was on the outer side of the right eye over the external rectus muscle. It was congenital, but had been growing lately. The child was then six years old. It was 7×3 *mm* and 3 *mm* thick, and movable on the sclera. It was made up of fibrous bands whose interstices were filled with fat cells, but the greater part of the growth was fibrous. BURNETT.

SHIRLEY (114) relates that he had occasion to scarify freely the conjunctiva of an apparently healthy negro infant of two weeks for muco-purulent conjunctivitis, and that the hemorrhage that ensued went on, in spite of all efforts to control it, to a fatal issue. BURNETT.

According to FUCHS (115) the pterygium develops from the pinguecula. It is covered with conjunctival epithelium, which may extend beyond its margins. Where the pterygium rests on the cornea, Bowman's membrane has mostly disappeared. But there are also changes in the corneal tissue in front of the anterior margin of the pterygium, consisting in the destruction of Bowman's membrane and in the loosening of the epithelium and the superficial lamellæ, proving that an affection of the cornea has preceded the development of the pterygium. The relation of the spreading margin of the pterygium to the cornea is not constant; at times it pushes as a loose, vascular tissue between the superficial lamellæ, at times it consists of a dense sclerotic connective tissue resting upon the normal Bowman's membrane. The first relation is found in progressive pterygia, the second in stationary. Fuchs believes that a pinguecula causes nutritive changes in the cornea. The superficial lamellæ of the cornea become loosened and separated, and the connective tissue of the limbus grows in. Since the nutritive fluids flow from the marginal network to the centre of the cornea, the disease of the cornea extends in this direction, and, in consequence, the pterygium grows toward the centre.

CLAIBORNE (116) burns off the excessive granulations in trachoma with the actual cautery, and completes the reduction of the tissue by applications of the copper stick. The acute stage he treats with ice applications and mild antiseptic washes.

BURNETT.

In WOLFNER'S (117) opinion all strong medication is out of place in treating conjunctival inflammations. He treats acute cases as he would a coryza, viz., hot foot-bath, warm drinks, rest in bed, and a cooling lotion to the eyes. The same general principle applies to purulent conjunctivitis, except that the eye must be kept clean. He opposed all astringents, and only weak germicides should be used. BURNETT.

118. HESS. Contribution to the study of filamentous keratitis. *v. Graefe's Arch.*, vol. xxxviii.

119. GILLET DE GRANDMONT. The microbic nature of keratitis. *Arch. d'Opht.*, vol. xii., 3, p. 149.

120. WILLIAMS, R. On the treatment of spreading ulcer of the cornea with hypopyon. *Lancet*, Jan., 1892.

121. DE WECKER. Corneal antisepsis. *Arch. d'Opht.*, vol. xii., 4, p. 201.

122. SIMI. Macula of the cornea. *Boll. d'Ocul.*, vol. xiv., 4.

123. COLBURN, F. E. Keratitis bullosa. *Amer. Jour. of Ophth.*, No. 4, 1892, p. 101.

124. CAPEI. A rare case of grave bilateral ocular lesion from lymph stasis in the cornea. *Boll. d'Ocul.*, vol. xiv., 10.

125. ADAMÜCK, E. On the use of sutures in corneal wounds. *Wjest. Ophth.*, vol. ix., 1, p. 16.

126. SIMON, R. A case of corneal neoplasm. *Centrabl. f. prakt. Augenheilk.*, 1892, p. 193.

127. GULLSTRAND, A. A case of keratoconus with rhythmic pulsation of the cornea. *Nord. Ophth. Tidskrift.*, vol. iv., 3, p. 142.

In the cases of filamentous keratitis that HESS (118) examined, the corneal epithelium played an important part in the formation of the filaments. The development of these is probably favored by a peculiar affection of the epithelium. It is not impossible that, besides the epithelium, at times also subepithelial tissues

take part in the formations, and fibrinous or mucous threads from the conjunctiva may adhere to the corneal filaments already formed.

GILLET DE GRANDMONT (119) attributes the development of corneal ulcers to micro-organisms which enter from without through some solution of continuity. Inoculating bits of the floor of an ulcer on agar-agar, gave cultures of various microbes, principally staphylococci, whose re-inoculation again produced ulcers. V. MITTELSTÄDT.

WILLIAMS (120) has obtained good results from the local application of quinine (4 gr.: ℥ j) in hypopyon ulcers. For paracentesis he prefers a radial incision in the sound part of the cornea. WERNER.

According to DE WECKER (121) most corneal diseases are of parasitic origin, and demand a corresponding treatment. The cause of the long course of the disease and the unsatisfactory results of treatment are due to the lack of a sufficient antisepsis. In order to bring active antiseptics as long as possible in contact with the affected spots, or to protect cautery wounds from a second infection, repeated instillations and a protecting bandage are not sufficient. De Wecker recommends his glass vessels, which may be laid on the anterior surface of the eyeball without causing irritation, and which may be used even in children. The antiseptic fluid is introduced through the central opening in the vessel, and may be left as long as necessary in contact with the eye. V. MITTELSTÄDT.

SIMI (122) has found in citric acid a means of removing maculæ and even leucomata of the cornea. The drug in concentrated solution is pencilled on daily. After two weeks, in place of the spot there is a depression which is allowed to undergo the usual regenerative process. As soon as the new tissue has reached the level of the corneal surface, the pencilling is repeated, and this is done at intervals until the spot disappears. In this manner the author was able to clear up, within nine months, an opacity for which in the beginning he had intended to perform an iridectomy. DANTONE.

COLBURN (123) has seen two cases of keratitis bullosa, one of which caused such destruction in spite of all treatment, as to lead to enucleation. The other healed after thorough cleansing of the vesicle and the application of 4 per cent. solution of arg. nitr.

He also reports three cases of pemphigus corneæ which subsided under evacuation of the vesicles and the pressure bandage.

BURNETT.

CAPEI (124) describes a case of bilateral lymph infiltration of the cornea, such as was first described by Rampoldi. The patient was a weak and poorly nourished peasant who worked with his head bent down. The finely granular and star-shaped opacity was so thick that the patient could only perceive movement of the hand. The remaining parts of the eye appeared normal. Under a strengthening general treatment and the local use of eserine, followed later by atropine and daily massage of the cornea, the disease improved and finally got well. A rapid increase in the opacity was noticed repeatedly while he was under treatment. The opacity was always denser in the morning than in the evening. When the patient bent his head down for fifteen minutes, the infiltration increased markedly, particularly in the centre of the cornea. DANTONE.

ADAMÜCK (125) regards the corneal suture as useless in corneal wounds, operative or traumatic. It is indicated, however, in traumatic defects of the cornea. When the apex of a keratoconus is removed, the round gaping defect heals much more quickly and completely after suturing. HIRSCHMANN.

SIMON (126) gives a description of the microscopic appearance of a corneal tumor. It was a fibroma with foci of myxomatous degeneration.

GULLSTRAND (127) described a case of kerato-conus in which a marked pulsating movement of the corneal images could be recognized with the ophthalmometer. Near the apex of the cone there was a systolic enlargement of the keratoscopic circles, nearer the base there was a systolic diminution of the circles, thus showing a tendency in systole to make the deformity uniform.

SCHIÖTZ.

128. NORSA. The medicamentous electric eye bath in the treatment of scleritis and episcleritis. *Arch. f. Augenheilk.*, xxiv., p. 186.

NORSA (128) found that the use of the constant current by means of a medicamentous fluid (lith. salicyl.) soon improved and eventually cured the affection.

129. PRAGER, C. A case of cysticercus cellulosæ in the anterior chamber. *Wiener klin. Wochensch.*, No. 14, 1892.

Sections XII.–XXI. Reviewed by Dr. P. SILEX.

Translated by Dr. CASEY A. WOOD.

XII.—IRIS.

130. BETTMAN, B. Traumatic Iridodialysis. *The North Am. Practitioner*, Dec., 1891.

131. HESS, C. A contribution to our knowledge of iridodialysis not of traumatic origin. *Zehender's klin. Monatsbl. f. Augenheilk.*, vol. xxx., p. 103.

132. HESS, C. A contribution to the study of iris colobomata in unusual situations. *Zehender's klin. Monatsbl. f. Augenheilk.*, vol. xxx., p. 106.

133. FERGUSON, H. L. Operation on microphthalmic eyes. *Oph. Review*, vol. xi., p. 43.

134. BADAL and FELIX LAGRANGE. Primary carcinoma of the ciliary processes and body. *Arch. d'Opht.*, vol. xii., No. 3, p. 143, 1892.

135. MITCHELL, S. Spasmodic mydriasis a sexual neurosis. *Am. Jour. of Ophth.*, Jan., 1892.

136. KLEBS. Remarks concerning von Donitz's communication on the cure of ocular tuberculosis. *Deutsch. med. Wochenschrift*, No. 50, p. 1358.

HESS (131) observed in a fourteen-year-old child a symmetrically placed iridodialysis without apparent changes in the sclera or cornea.

HESS (132) found a bilateral coloboma of the iris directed inwards and upwards; also a downward choroidal coloboma on the right side, accompanied by other alterations in the eye. "The embryonic connective tissue from which the vitreous and fœtal lens sheath are formed was abundantly and abnormally developed. The incomplete or too late development of these presented on the one hand a complete closure of the optic cleft; on the other it disturbed the growth of the iris by delaying the formation, inward and upward, of the vessel-bearing lens sheath."

FERGUSON'S (133) first case had an iridectomy performed in the left eye for glaucoma. The nerve was atrophied and V= hand-reflex. The right eye had V = $\frac{6}{60}$ with + 18 D. The refraction was 27 D. Both eyes were small and the cornea meas-

ured 10 *mm* horizontally, and 0.5 *mm* less vertically. In the second case the corneæ measured about 7 *mm* in diameter. There were extensive posterior synechiæ and opaque lenses. After several iridectomies and needlings V = fingers at 3 *m*. WERNER.

BADAL and LAGRANGE (134) noticed, in a boy eight years of age, a neoplasm of the ciliary body which appeared to have developed shortly after birth. At the age of five the eye had become blind, and when enucleation was later on performed, there was found an intercalary, ciliary staphyloma of the size of a bean; pupil widely dilated; anterior chamber deep; cornea intact; fundus invisible. The tumor was the size of a pea, with a projection from it one third as large; it touched the ciliary region and stretched to the periphery of the shrivelled lens. It presented all the appearances of a glandular carcinoma, and consisted of numerous naked tubules provided with a number of single-nucleated cylindrical cells. The former were partly filled with proliferating cells that had infiltrated the neighboring tissues. The normal elements of the ciliary body had been destroyed and very few vessels and but little pigment could be found. The discovery of a glandular cancer in the *pars ciliaris retinæ* seems to support Nicati's theory of a *glande de l'humeur aqueuse.*

v. MITTELSTÄDT.

MITCHELL (135) has had under observation five cases of spasmodic intermittent mydriasis, two men and three women, in which he believed the condition was brought about by excessive venereal excitement. BURNETT.

XIII.—CHOROID.

137. RINDFLEISCH, G. Experimental examination of the decreased tension attending purulent choroiditis. *Graefe's Archiv. für Oph.*, vol. xxxviii., 2, p. 221.

138. PHILLIPSEN, H. Et Pas Notitser of oftalmologisk diagnostisk. *Inhold. Hosp. Tidende*, S. 783 et 868, Kjöbenhaven, 1891.

139. RUMSCHEWITSCH, K. On some doubtful points connected with pseudo-glioma of the retina. *Zehender's klin. Monatsbl. f. Augenh.*, vol. xxx., p. 230.

RINDFLEISCH (137) found, immediately after the introduction of metallic mercury into the vitreous, increased tension accompanied by marked cloudiness of the retina; then reduction of the

tension below normal during the commencing vitreous cloudiness, and its successive diminution as the latter progressed to the stage of suppuration. At the same time the intraocular pressure was found to be plus in the non-injected eye, and it so remained while the affected eye went on to *phthisis bulbi.* The anatomical examination threw no light on the production of the tension.

PHILLIPSEN (138) asserts that partial paralysis of the iris (on the side where the tumor is situated) with good visual acuity is pathognomonic of commencing choroidal sarcoma. SCHIÖTZ.

[This symptom is not frequent in the early stage.—H. K.]

RUMSCHEWITSCH'S case (139) was clinically one of retinal glioma, but after removal of the eye he found an inflammation of the choroid, complicated by detachment of the retina.

XIV.—GLAUCOMA.

140. KABINOWITSCH, G. Another instance of the efficacy of iridectomy in a case of glaucoma attended by complete loss of light perception. *Wjestn. Ophthalm.*, 1892, May to June.

141. DE SCHWEINITZ, G. E. Coloboma of the iris, polycoria, and primary glaucoma. *Trans. Amer. Ophth. Soc.*, 1891.

142. SCHNABEL. Glaucomatous affections of the optic nerve. *Archiv f. Augenh.*, vol. xxiv., part 4.

143. GARNIER, R. Remarks upon the endarteritic changes in the ocular vessels. *Centrabl. für prakt. Augenheilk.*, vol. xvi., p. 9.

144. GARNIER, R. Some observations concerning glaucoma, and the vascular changes associated with and dependent upon it. *Archiv f. Augenh.*, vol. xxv., p. 24.

145. ULRICH, R. The experimental production of glaucoma in rabbits. *Arch. f. Augenh.*, vol. xxv., 1st and 2d part, p. 2.

146. POMEROY, O. D. A report of cases of glaucoma of different varieties, illustrating some of the usual means practised for their relief. *N. Y. Med. Jour.*, March 19, 1892.

KABINOWITSCH (140) saw a case of bilateral glaucoma where perception of light had been completely lost for eight days. Double iridectomy. After five weeks, V R = $\frac{18}{30}$; V L = $\frac{18}{200}$; restriction of the visual field above, in, and below.

In DE SCHWEINITZ'S case (141), in addition to the unusual feature of an attack of glaucoma in an eye affected with coloboma

of the iris, for which an iridectomy was successfully done—as regards these symptoms—the deformity itself is peculiar in that, besides the coloboma itself, there are three other distinct openings in the iris tissue upward and outward. There are also two other places where the iris is very much thinned. BURNETT.

SCHNABEL (142) holds the opinion that the excavation in glaucoma is produced by a retrobulbar neuritis and atrophy of the intraocular portion of the opticus. He believes that not only the fibre-nerves themselves, but all the constituents of the opticus, undergo atrophy. In proof of this, he cites a case where (with one half vision and an almost complete field) there was an advanced atrophy of the nerve-fibres in the non-medullated portion of the opticus. The nervous framework was also affected, and in the medullary segment a chronic interstitial neuritis in various stages was found to extend from behind the lamina cribrosa to the chiasma. That this was an undoubted case of glaucoma is by no means proven. Schnabel has not made allowance for the fact that the ophthalmoscopic picture of optic-nerve atrophy with a pre-existing physiological excavation cannot be distinguished from that of glaucomatous cupping. SCHWEIGGER.

GARNIER (143) speaks of the beaded and diffuse compensatory endarteritis described by Thomas as occurring in many ocular diseases—especially glaucoma. This consists, at the outset, of degenerated hyaline or connective-tissue-like masses between the elastic membrane, and shows itself in those cases where in consequence of the decreased elasticity of the middle muscular coat with secondary local enlargements of the vascular lumen a slowing of blood current results. Since it is especially in cases of glaucoma that these conditions are present, and where, too, the capillaries are much more permeable than normal, slight external causes may bring about increased transudation followed by increase of ocular tension. HIRSCHMANN.

ULRICH (145) produced, by the gradual removal of the cornea of rabbits at several sittings, total anterior synechia. The iris became thickened, fibrous, and atrophic. The ciliary processes were hyperæmic, and showed isolated pigmentary deposits. The congestion followed from the hypersecretion ; indeed, a glaucomatous outbreak showed itself as soon as the cicatricial tissue became firmly attached and made the normal filtration of the fluids no longer possible. As a rule, this happened when the altered iris became the subject of fibrous degeneration at its circumference.

The incarceration of the iris tissue acted directly and the fibrous degeneration and atrophy were the indirect agents in producing a disturbance of the blood supply of the iris. The latter, in its turn, brought about a secondary, collateral congestion of the ciliary processes, thus furnishing evidence, so far as such instances of glaucoma are concerned, in support of the hypersecretion theory.

POMEROY (146) gives full notes of twenty cases (32 eyes) of glaucoma of all forms. Iridectomy was the sheet anchor in treatment, but eserine has proven of great value, generally in solution of 1 gr. to ℥ j, sometimes 2 grs. Paracentesis corneæ has proven useful in temporarily relieving pain. Sclerotomy has also succeeded in relieving pain when iridectomy has failed.

BURNETT.

XV.—SYMPATHETIC OPHTHALMIA.

147. BULLER, F. Conservative surgery of the eye. *Ophth. Rec.*, Jan.-Feb., 1892.

148. JOHNSON, G. LINDSAY. Note on two cases of obscure sympathetic phenomena. Translated from the original manuscript by H. Parent. *Arch. d'Ophtalmologie*, vol. xii. No. 1, 1892, p. 51.

149. SCHMIDT-RIMPLER. Contribution to the etiology and prophylaxis of sympathetic ophthalmia. *Graefe's Arch. f. Ophth.*, vol. xxxviii., 1, p. 199.

While believing that the sympathetic ophthalmia which occurs after the offending eye has been removed is much less virulent than that which occurs when the enucleation has been too long delayed, BULLER (147) nevertheless thinks that the eye of a child should never be enucleated on account of an injury unless sympathetic ophthalmia has actually occurred. He thinks resection of the optic nerve prevents sympathetic ophthalmia oftentimes, and in suitable cases should always be tried before enucleation is finally resorted to. He also practises evisceration, particularly where the injury is confined to the anterior part of the eye.

BURNETT.

G. LINDSAY JOHNSON (148) examined a waiter, æt. 42½ years, who, for 2½ years after an enucleation, wore a glass ball 2 *cm* in diameter within his conjunctival sac. The other eye, until that time healthy, showed these symptoms: transitory ciliary injection, weakness of accommodation, severe pain on pressure over the

glass globe, not only in the corresponding orbit, but *at the same time behind the sound eye.* V of the latter normal; J. i. read with hesitation; F of V considerably and to a variable extent constricted; fundus normal. After removal of the artificial vitreous all these symptoms disappeared.

The second case was that of a lady, aged 39, who has been frequently treated for repeated attacks of iritis associated with opacities of the cornea. *The corneæ were so cloudy* ("les deux cornées étaient infiltrées de taches si étendues, qu'elles n'étaient transparentes qu'en deux points") that V, left, = fingers at 1 *m*, and right at 2 *m*. Four days after an iridectomy *on the right eye*, the cornea of the *left* was almost clear, and a proposed iridectomy seemed needless, V = $\frac{5}{6}$ and, J. i. Author had carefully examined these rare cases with other *confrères*, and is undecided whether the disease should be regarded as a *sympathetic* keratitis or not.

V. MITTELSTÄDT.

SCHMIDT-RIMPLER (149) mentions the following peculiar cases: 1. Optico-ciliary neurectomy; sympathetic ophthalmia after 1½ years. Demonstration of normal nerve fibres in the ciliary body of the enucleated stump. 2. Sympathetic ophthalmia without premonitory tenderness on pressure. The first symptom was iritis serosa; then increase of the inflammation due to trauma of the previously injured eye. Recovery. He discards the migration theory, not only because of negative results from experiments, but also on account of the evidence furnished by clinical, morbid-anatomical, and bacteriological investigations. The ciliary-nerve theory appears to him more plausible. He sees in the irritation of the ciliary nerves of the wounded eye (causing a reflected disturbance of the blood circulation and nutrition of the other eye) the single factor in the predisposition to sympathetic inflammation of the opposite organ. In this way the ground is prepared for those influences, be they bacterial or chemical, which light up inflammation. In spite of this opinion he does not reject resection of the optic nerve as a prophylactic measure. When, however, the disease has proclaimed itself, the eye should always be excised.

XVI.—VITREOUS.

150. WAGENMANN. Further communications regarding vitreous suppuration from the scars of iris incarcerations. *v. Graefe's Archiv für Ophthalm.*, Bd. xxxviii., p. 171. (Cases of infection from without through the medium of the cicatrix.)

XVII.—LENS.

151. MAGNUS, H. Ophthalmological charts for academical teaching and private study. II., The development of senile cataract. Breslau, 1892.

152. EWETZKY. Cataract and zerosis of the cornea in glass workers. *Deutsche med. Zeitung*, 8th Oct., 1891.

153. FUCHS, E. On lenticular precipitates. *Beiträge zur Augenhlk.*, pt. 3, 1891.

154. MITVALSKY. A recent case of lenticonus posterior with partial persistence of the hyaloid artery. *Centralb. f. pkt. Augenheilk.*, xvi., p. 65.

155. TOPOLANSKI, A. Irregularities of the lenticular border. *Zehender's klin. Monatsbl. f. Augenhlk.*, vol. xxx., p. 89.

156. RYERSON, G. S. Miscellaneous facts regarding the extraction of cataract. *Ophthalmic Record*, Jan.-Feb., 1892.

157. PFLÜGER. Observations on the present state of cataract extraction. *Zehender's klin. Monatsbl. für Augenheilk.*, vol. xxx., p. 155.

158. SCHULECK, W. A new method of cataract extraction. *Pester med.-chir. Presse*, No. 23, 1892.

159. WAGNER. Report of 1,000 extractions with small peripheral flap (von Graefe's method). *Wjestnik Ophthalm.*, 1892, No. 1.

160. DOLSCHENOW. Review of 400 cataract extractions. *Wjestnik Ophthalm.*, 1892, No. 1.

161. WEBSTER, DAVID. Report of 136 cases of cataract extraction with remarks. *Trans. Am. Oph. Society*, 1891.

162. LIPPINCOTT, J. A. Routine syringing out of cortical substance in cataract extraction as illustrated in 100 cases. *Trans. Am. Oph. Society*, 1891.

163. ARNOLD, TH. Report of 400 cataract extractions performed by Prof. O. Haab on patients in the Zurich University clinic and in his private hospital. *Arch. für Augenheilk.*, Bd. xxv., Pts. 1 and 2, p. 41.

164. HIRSCHBERG, J. On extraction of nuclear cataract. *Berlin. klin. Wochenschrift*, 1892, p. 635 ; *Sem. Centralblatt.*

165. HJORT. Cataract extraction without iridectomy. *Norsk Mag. f. Laegerid, Christiania*, 1891, p. 643.

166. KRAMSZTYK. Fate of the iris after cataract extraction. Congress of Polish physicians and naturalists, 1891. *Zehender's klin. Monatsbl. f. Augenheilk.*, vol. xxx., p. 294.

167. SIMI. Suture of the cornea after extraction of cataract. *Boll. d'Ocul.*, vol. xiv., p. 1.

168. BULLER, F. Glaucoma after cataract extraction. *Trans. Am. Oph. Soc.*, 1891.

169. KEYSER, P. D. Remarks on extraction of cataract. *Annals of Oph. and Otol.*, Jan., 1892.

170. WOOD, CASEY A. A contribution to the study of concussion cataract. *Ann. of Oph. and Otol.*, Jan., 1892.

171. KNAPP, H. The occurrence, prevention, and management of prolapse of the iris in simple extraction of cataract. *Trans. Amer. Oph. Soc.*, 1891.

172. THEOBALD, S. Supplementary note to the case of useful vision maintained by the aid of a totally dislocated lens, heretofore reported to the society.

173. ROOSA, ST. JOHN. The best operation for the extraction of cataract. *Ann. of Oph. and Otol.*, Jan., 1892.

174. RANDOLPH, R. L. A series of fifty consecutive operations for cataract. *Johns Hopkins Hosp. Bull.*, No. 20, March, 1892.

MAGNUS (151), in a series of eleven plates, illustrates the development of cataract in a manner that might be expected from his numerous recent and well known studies of the subject. In a few minutes one can be thoroughly informed upon everything pertaining to this matter.

EWETZKY (152) lays the blame upon the very great heat encountered during the work.

FUCHS (153) means by "lens precipitates" dotted markings of a grayish-brown color, on the inner wall of the cornea, that are associated with swelling of the lens. The aqueous humor contains much fibrin and pigment cells derived from the posterior surface of the iris. Absorption takes place readily.

TOPOLANSKI (155) states that the lenticular border is not always smooth but is frequently wavy and, particularly above, presents cone-shaped projections.

As a rule RYERSON (156) closes his extractions with an iridectomy, but lately has been trying the simple method. He uses eserine after the operation, but in weak solutions. In some cases

he washes out the anterior chamber with Panas' solution. He uses a knitted Liebreich bandage. Has abolished dark rooms and does not confine his cases to the bed. He has operated successfully on eight patients between eighty and ninety years of age. His extractions number 250. BURNETT.

PFLÜGER (157) replies at length to questions propounded by Landolt. He is such a firm believer in both asepsis and antisepsis that he operates, when at all possible, without iridectomy. He does not consider that every cataract after the sixtieth year is fit for extraction. Washing out the anterior chamber is, to his mind, a valuable adjunct to the technique, and for the purpose he uses a boric solution or trichloride of iodine 1:4000. Discission wounds more readily tend to infection than others. Shreds of vitreous hanging out of the wound of incision are best treated by touching them with the galvano-cautery.

WAGNER'S (159) 1,000 cases of cataract operation are divided into three classes: (1) 474 extractions without antiseptics, with 8.86 per cent. of losses. (2) 223 extractions under strict antiseptic precautions, but performed, like the first group, in general hospitals, with 5.82 per cent. of loss. (3) 303 extractions, antiseptic, but made in the well arranged eye hospital of Odessa, with but 2.43 per cent. of failures. Before practising Graefe's method, which Wagner still employs in all his cases, complicated and uncomplicated (his statistics include all cases without exception), he made the classical flap operation and had 16 per cent. of failures. The first group furnished 16 discissions and 3 extractions of secondary cataract, 19 iridectomies and 18 iridotomies; the second, 6 discissions and two extractions of secondary cataract, 5 iridectomies and 6 iridotomies; the third, 2 discissions, 2 secondary extractions, and 2 iridectomies. Wagner is opposed to extraction without iridectomy and to extractions with the capsule entire. Wagner regards the use of the spring speculum and the fixation forceps as indispensable up to the moment of the removal of the lens. Many years of experience have convinced Wagner of the superiority of the Graefe method; he would still practise the preliminary iridectomy when, all things considered, it is permissible.

In making extraction by the simple method WEBSTER (161) tries to include a little less than half of the circumference of the cornea and endeavors to get a small conjunctival flap. He uses eserine in weak solution after the operation.

The eye is not examined for forty hours. In 116 simple extractions there was 91 per cent. of good results, 5 per cent. of medium, and 3 per cent. of failures. In 20 cases by "Gräfe's method" there was 75 per cent. good, 15 per cent. medium, and 10 per cent. of failures. In the simple extractions prolapse occurred in 4 per cent. of the cases. He believes in early excision in cases of prolapse. BURNETT.

LIPPINCOTT (162) makes a preliminary iridectomy generally downward.

The incision, in extraction, lies wholly in the cornea and embraces $\frac{1}{8}$ to $\frac{1}{2}$ of the corneal circumference. The lens is delivered by pressure of the thumb against the upper-lid edge at the upper margin of the cornea, and the anterior chamber washed out by means of a syringe of his own devising, with a solution of boric acid 2 to 4 grs. to $\frac{\mathfrak{z}}{3}$ j. A quantity varying from 2 drachms to 2 or 3 ounces is allowed to pass in and out of the anterior chamber.

All the cases are tabulated. Two cases had occluded pupils and one case was lost after the preliminary iridectomy, from suppurative panophthalmitis, though the operation was done under strict antisepsis. The author's anterior chamber syringe is figured. BURNETT.

After a careful statistical examination in reference especially to the operative procedure, the eventual visual acuity, complications, secondary operations, etc., ARNOLD (163) concludes that the combined operation is to be preferred to the simple method. Cases of lamellar cataract are never treated by optical iridectomy.

HIRSCHBERG (164) reports over 200 cases. 75 obtained V of more than $\frac{1}{2}$; 10 per cent. had diabetes.

Prof. HJORT (165) prefers extraction without iridectomy and believes that by a modification of his former method of placing the corneal incision the danger of iris prolapse is lessened. Previously the highest point in the corneal flap never approached nearer than $\frac{3}{4}$—1 *mm* to the limbus; now he makes a cut parallel with the plane of the iris so that it lies entirely within the transparent margin of the cornea and often forms a small conjunctival flap. SCHIÖTZ.

SIMI (167) objects to Suarez' method of suturing the cornea after cataract extraction. The writer would feel inclined to resort to the suture only in those exceptional cases where, owing to the restlessness of the patient, there is danger of rupturing the

recently healed wound. In such instances he prefers the scleral stitch of Barramer. DANTONE.

BULLER (168) has seen glaucoma supervene in three eyes after cataract extraction. In two it came on some years after the operation, and was of the simple non-inflammatory type. In the other it was of the sub-acute variety and eserine had a beneficial effect. He thinks that excessive use of the eyes may have had some effect as a cause. BURNETT.

KEYSER (169) has had as good success in extracting unripe cataracts as ripe ones, and pays special attention to the manner of dividing the anterior capsule. He usually makes a T-shaped incision, but thinks removal of a piece by means of Wecker's forceps good. For ordinary ripe cataracts he thinks simple extraction the better method. BURNETT.

WOOD (170) is inclined to consider some cases of cataract which come on very soon after concussion, of which he relates the history of two, as due to a physical displacement of the molecules of the lens-substance. BURNETT.

KNAPP (171) regards iris prolapses as the weak point in simple extraction, and one against which it is most difficult to guard. Its frequency of occurrence varies in his own statistics from 6 per cent. to 12 per cent. For its prevention he advises: (1) To make an iridectomy when, in the particular cases, all points considered, the combined method offers better chances for restoration of sight than simple extraction; (2) cocainize thoroughly; (3) make the corneal incision in the classical way of Beer; (4) cleanse the anterior chamber thoroughly; (5) open the capsule by one long horizontal incision in the upper part under the iris; (6) avoid prolapse of vitreous and everything that ruptures the transverse septum of the globe; (7) carefully replace the iris; (8) myotics may be used; (9) keep the patient as quiet as possible for the first day or two; (10) watch the patient or tie his hands during the night for the first five or six days. After prolapse has occurred let it alone for several weeks. If it should not diminish by that time abscise. Early abscissions, except in the first twenty-four hours he does not think wise. BURNETT.

THEOBALD (172) reports in regard to a case of dislocated lenses—that ten years afterward in the left eye the lens had become cataractous and finally the capsule ruptured, the contents being diffused into the vitreous with symptoms of glaucoma. Under treatment these symptoms disappeared and the vitreous

cleared up, and vision was restored with glasses to the former state of $\frac{1}{1}\frac{1}{8}$. The lens floating in the vitreous of the other eye is intact and gives no inconvenience. BURNETT.

ROOSA (173) prefers the simple extraction for straightforward cases, reserving the linear operation and preliminary iridectomy for exceptional cases. He uses eserine after the extraction in the simple operation and still adheres to the flannel bandage.

BURNETT.

The principal interest in this series of operations (174) attaches itself to the fact that the operations were done for the most part in the cabins of negroes in the country or under other unfavorable conditions. An iridectomy was made, as a rule. Two eyes were lost—one from internal hemorrhage and one from corneal suppuration. BURNETT.

XVIII.—RETINA AND FUNCTIONAL DISTURBANCES.

175. ROSENTHAL. Case of snow-blindness. *Hygeia*, p. 299, Stockholm, 1891.

176. AYRES, S. C. Retinitis pigmentosa in the colored race. *Amer. Jour. Ophth.*, March, 1892.

177. BULL, C. S. Operative treatment of retinal detachment by Schöler's method, with a report of five cases. *Trans. Amer. Ophth. Soc.*, 1891.

178. SCHOENFELD, W. Contributions to the treatment of retinal detachment. *Inaug. Dissert.*, Berlin, 1892.

179. ALEXJEW. A case of hemorrhagic retinitis in progressive pernicious anæmia.

180. BJERRUM, J. Et tilfeede af embolia art. centr. retinæ med. et ejen dommaligt kredslöbsfænomen. *Nord. Ophth. Tidsk.*, vol. iii., p. 270, Kjöbenhavn, 1891.

181. PERLES, M. Clinical studies of vascular disease of the eye. On partial embolism of the retina. *Centralbl. f. prakt. Augenhlkde.*, vol. xvi., p. 161.

182. LÖWENFELD, L. The objective signs of neurasthenia. *Münch. med. Wochenschrift*, No. 50, p. 856.

183. WILBRAND and SÄNGER, A. Further observations on visual disturbances in functional diseases. *Jahrb. der Hamburger Staats-Krankenanstalten*, 1890, part 1.

184. KÖNIG. On fatigue of the retina and its connection

with concentric restriction of the visual field in diseases of the central nervous system. *Neurol. Centralbl.*, 1892, p. 449.

185. GRAEFE, A. Note on the use of the prism in detecting simulation of unilateral amaurosis. *Zehender's klin. Monatsbl. f. Augenhlkde*, vol. xxx., p. 260.

186. HEYL, A. J. The calibre of the retinal vessels after traumatic injury to the convexity of the brain. *Trans. Amer. Ophth. Soc.*, 1891.

187. SUTPHEN, T. Y. Amaurosis in women. Three clinical cases. *Trans. Amer. Ophth. Soc.*, 1891.

AYRES (176) relates the history of a case of retinitis pigmentosa in a negro man of forty-five. [The reviewer has seen retinitis pigmentosa in the negro race a number of times.]

BURNETT.

BULL (177) gives the history in detail of five cases of detached retina which he treated by Schoeler's method of injection of tincture of iodine after other methods had failed. In none were any permanent good results obtained. BURNETT.

SCHOENFELD (178) shows from a study of the literature of the subject, and from a history of twenty-three patients treated in the University Clinic in Berlin by Schœler's method of injecting iodine that no advance has thus been made in the treatment of retinal detachment.

Although retinal hemorrhage is common enough, its occurrence with progressive anæmia is so rare that ALEXJEW (179) can find only three cases in literature.

In a case of embolism of the arteria centralis BJERRUM (180) noticed (in an artery) during the first two or three days a rapid and continuous current in a *centripetal* direction. The blood was very dark and contained small clots; later on it presented the ordinary bright-red color of the remaining arteries.

SCHIÖTZ.

PERLES (181) reports six cases. The hope of regaining central vision is well founded when there is an uninterrupted current in the affected arteries or when they pulsate on bulbar pressure. Treatment consists of faithful and energetic massage under cocaine.

WILBRAND (183) desires to replace with the term nervous asthenopia the name neurasthenic asthenopia = anæsthesia retinæ = peripheral amblyopia of the visual field, formerly em-

ployed by him. He includes in it all the various symptoms that accompany functional disturbances of the nervous system. He discusses the nervous asthenopia of school children, that occurring during the developmental period, that seen in neurasthenic adults, and especially that noticed in hysterical persons. Lastly he refers to the neuroses occurring in traumatic cases.

Of 215 patients examined by KÖNIG (184) in the Dalldorf Lunatic Asylum, 81 had defects of the visual field. They all showed the Förster-Wilbrand "fatigue" symptoms (the examination being first made in the horizontal meridian) and a symmetrical concentric contraction. The observations of others were confirmed that the fatigue is most marked at the commencement; that the temporal side is more readily tired out than the nasal, and that in some instances the temporal side alone is thus affected. Of interest is the description of one case of the so-called "oscillating field" (partial disappearance of the perimetric object) which had lasted one and a quarter years. Simulation of a regular limitation of the visual field, with or without the "fatigue" symptom, is said to be impossible.

GRAEFE (185) recommends that the diseased eye be covered while a prism of 18°, base down, is placed below the sound eye and slowly raised in front of it. At a certain point the patient has diplopia and learns that there is nothing suspicious about double images. The other eye is now opened and the experiment repeated in such a way that if he sees two images one can be sure that the alleged blind eye is really capable of vision.

The gist of HEYL'S (186) paper and the histories of the cases which he cites (twelve in number) tend to show that there is an immediate connection between the calibre changes in the retinal vessels and delirium and sopor alternans. The retinal changes, however, he thinks probably indicate a corresponding change in the large intracranial trunks. The relations of such change, however, are unknown. BURNETT.

SUTPHEN'S (187) cases are: (1.) A healthy girl of eighteen was driven out-of-doors bare-foot and in her night dress, and walked in the snow a quarter of a mile during her menstruation. Eight weeks after, vision in right eye began to fail. Ophthalmoscope failed to reveal any adequate cause for the blindness except that the vessels seemed smaller than normal—fingers at two feet. Some weeks later the left eye went suddenly, entirely blind. (2.) Six weeks after confinement a woman was suddenly seized with

severe headache, and vision in the right eye was lost. Two weeks later the other eye became suddenly blind. Vision became at times somewhat better but finally it remained p. l. in L. and fingers at ten feet in R. Discs pale and signs of atrophy. (3.) A case of blindness in one eye lasting three months, followed by improvement, without any cause, local or general.

BURNETT.

XIX.—OPTIC NERVE.

188. BEER. On a case of optic-nerve disease in *uterus infantilis. Wien. klin. Wochenschrift*, 1892, Nos. 30, 31. (Retrobulbar neuritis; finally full visual acuity with pallor of the papillæ.)

189. OTTO. On changes in the optic nerve, particularly in cases of arterial sclerosis. *Centralb. f. Neurologie*, S. 447, 1892.

190. LEBER. Peripheral optic-nerve affections in hysterical subjects. *Deutsche med. Wochenschr.*, 1892, p. 741.

191. DEUTSCHMANN. On acute retrobulbar neuritis of rheumatic origin. *Beiträge zur. Augenheilk.*, pt. vi., p. 899.

192. GROENOUW. On toxic amblyopia. *v. Graefe's Archiv f. Ophthal.*, vol. xxxviii., 1., p. 1.

193. RYDEL. On the question of amblyopia as a result of alcohol and tobacco poisoning. Congress of Polish Physicians and Naturalists, 1891. *Zehender's klin. Monatsbl. f. Augenheilk.*, vol., xxx., p. 294.

194. DE SCHWEINITZ, G. E. Additional experiments to determine the lesion in quinine blindness. *Trans. Amer. Oph. Soc.*, 1891.

195. CALLAN, P. A. Two cases of orbital traumatism resulting in immediate monocular blindness through fracture into foramen opticum. *Trans. Amer. Oph. Soc.*, 1891.

196. TAYLOR, L. N. A case of brain tumor with high grade of choked disc; autopsy. *Trans. Amer. Oph. Soc.*, 1891.

197. HUBBELL, A. A. Optic neuritis as a form of peripheral neuritis. *N. Y. Med. Four.*, Jan. 22, 1892.

198. WOOD, CASEY A. The retrobulbar neuritis of young adults. *Ophthalmic Record*, June, 1892.

OTTO (189), in discussing the topography of the optic nerve behind the foramen opticum, describes some congenital malfor-

mations discovered by him. He also asserts that the mechanical influence of dilated and calcified vessels often produces organic changes. Certain forms of atrophy in old people are probably associated with these alterations.

LEBER (190) has observed a number of cases of hysterical amblyopia and amaurosis which were the outcome of a retrobulbar neuritis. In spite of rapid restoration of vision sometimes pallor of the disc showed itself later on. Of five cases four were women. This orbital neuritis, which may quickly get well without treatment, probably leaves behind it, according to Leber, a disturbance of optic conduction, which, in its turn, disappears or goes on to atrophy.

DEUTSCHMANN (191) describes seven cases in which no other cause than cold could be discovered. The disease probably began as a simple periostitis about the foramen opticum.

GROENOUW (192) presents the well-known clinical picture—defective vision, intact peripheral field with oval scotoma for red between fixation point and blind spot, unchanged light sense, etc. —and from an examination of 178 cases concludes that the disease is most common between the fortieth and fiftieth year, and its principal cause is tobacco. Only two cases could be charged to alcohol alone. In the obstinate cases recovery set in only after six months. The characteristic and constant symptom is a central defect for red. The combined extent of the scotoma in advanced cases measured 22° in the horizontal direction and 16° vertically. Worthy of remark are the cases of slowly progressive optic atrophy accompanied by peripheral limitation of the field. In the matter of differential diagnosis there is to be considered chiefly axial neuritis, which, however, besides perimetric defects in the form of re-entrant angles, shows central scotomata for red *and* white.

DE SCHWEINITZ (194) presented some photomicrographs made by Dr. Gray of the Army Med. Museum showing the effects upon the nerve tissue in artificial quinia poisoning made in experiments by De Schweinitz. The results seem to show that the effects of toxic doses of quinia are upon the whole visual path, including the optic nerves, chiasma, and tracts as far as they can be traced. The other nerves of the eye do not seem to be affected. The process probably begins by a constringing of the vessels through the vasomotor nervous system and finally inflammation is set up —probably an endovasculitis—in which thrombosis may occur

resulting in atrophy. The experiments were made mostly on dogs. BURNETT.

In CALLAN'S (195) first case, the point of a fencing foil entered the orbit between the nose and eyeball. There were symptoms of orbital cellulitis and the fundus was hazy and the nerve finally became white, though there is still some vision on the nasal side. In the second case the blow was over the left eye and blindness was said to have been immediate. There was, fifteen hours after the injury, no change observable in the fundus, but in a week the nerve began to grow pale, and in three weeks the atrophy was pronounced. BURNETT.

TAYLOR'S (196) case was a boy of seventeen. He had at first examination V = $\frac{1}{10}$ though there was a high degree of papillitis. His principal complaint was dizziness and persistent headache. Subsequently atrophy and blindness ensued. There was some exophthalmos particularly in right eye; some slight paresis, but no permanent paralysis. He died suddenly, and a tumor was found between the cerebrum and cerebellum, which arose from the corp. quad. and roof of the fourth ventricle. The tumor was a glioma. BURNETT.

HUBBELL (197) thinks that from analogy and also from direct evidence we are warranted in regarding optic neuritis as a form of peripheral neuritis and dependent upon general systemic conditions. BURNETT.

CASEY WOOD (198) reports two cases of retrobulbar neuritis, one a woman, the other a man, in both of whom there was remaining a perfect central scotoma for white, and a scotoma for colors extending 5° to 10° outside of this.

The fundus and nerve-head remained normal. He considers that usually such cases go on to recovery. BURNETT.

XX.—INJURIES, FOREIGN BODIES, AND PARASITES.

199. GOTTI. Two important cases of ocular traumatism. *Bull. d. Scienze mediche*, series vii., vol. iii.; and *Boll. d'Ocul.*, vol. xiv., p. 6.

200. STRAUB. Een tweetal verwondingen der oogholte en haar mechanisme. *Weekblad van het Nederlandsch Tijdschrift voor Geneeskunde*, vol. ii., p. 565, 1892.

201. RAMM. Enucleation of the eye; meningitis; death. *Norsk. Mag. f. Laegerid*, p. 502, Christiania, 1891.

202. ROOSA, ST. JOHN. Extraction of foreign bodies from the eyeball. *Trans. Am. Oph. Society*, 1891.

203. PRAGER. A case of cysticercus cellulosæ in the anterior chamber. *Wien. klin. Wochenschrift*, 1892, p. 209.

204. SEYFERT. Contributions to the operative treatment of intraocular cysticercus. *v. Graefe's Archiv f. Ophthalm.*, vol. xxxviii., 2, p. 112.

205. HIRSCHBERG, J. On trichinosis of the human eye. *Berlin. klin. Wochenschr.*, 1892, p. 325.

206. HOLT, E. E. Extraction of foreign bodies from the vitreous. *Trans. Amer. Oph. Soc.*, 1891.

207. SCHWARTZSCHILD, H. DAVIDSON. A case of palimptosis alternating with proptosis following injury. *Med. Record*, May 4, 1892.

In one of GOTTI'S *two* cases (199) of injury to the eye a large grain of shot entered the anterior chamber. Twenty-four hours afterwards not a single trace of the wound could be found. It was supposed that the blood in the anterior chamber resulted from simple contusion, and after the disappearance of the first signs of irritation the patient was discharged as practically cured. But after a year had passed severe symptoms suddenly manifested themselves. On incising the cornea an abundant aqueous exudation poured out and disclosed the presence of a No. 6 shot. On removing the latter the eye was again restored to health. The *second* case was that of a total detachment of the iris following a collision with the corner of a chair. The only sequelæ were three small scleral staphylomata and marked photophobia in a very bright light. DANTONE.

In RAMM'S case (201) the eye was destroyed by a penetrating wound from a splinter of wood. The foreign body had been removed and the wound healed with a total anterior synechia. When seen the eye was red, the conjunctiva chemotic, the globe hard, and there was much pain. The eye was excised under strict antiseptic precautions; instruments and absorbent cotton were sterilized by means of Arnold's steam apparatus; the parts were cleansed with sublimate solution, and the orbit was stuffed with iodoform gauze. There was a little pain the first evening, which disappeared with the removal of the tampon. Next day fever and delirium. Wound washed out with $\frac{1}{10}$ % sublimate solution. Death on the second day. Autopsy revealed purulent

infiltration at the base and convexity of the brain. No foreign body found in the enucleated eye. SCHIÖTZ.

ROOSA (202) reports three cases. (1) That of a bit of metal entangled in and removed from the tissue of the iris which had been drawn out through a corneal incision. The iris was replaced and the case did well. (2) A bit of thin wire 12 *mm* long was lodged in the lens near its equator and projected into both the anterior and vitreous chambers. The anterior chamber was opened by an incision and the wire seized by its point and pulled out. Recovery good. Patient could distinguish small objects at twenty feet. The third case was a foreign body—bit of steel—in the vitreous chamber, which was removed in the usual manner by the electromagnet. Vision was nearly normal. BURNETT.

SEYFERT (204) reports six cases operated upon in the Göttingen clinic between 1886 and 1889. In three of these permanent vision was secured, while all six globes were saved. Result similar to those obtained in the clinic at Halle.

HIRSCHBERG (205) treats exhaustively of every possible subject connected with tapeworm and cysticercus—history, developmental stages, channels of infection, duration of life, with statistics relating thereto,—and concludes that the strict inspection of meat (in Germany) has almost driven out the disease. In the course of the year he has treated 41 cases of trichinosis. In 32 instances where the parasite lay beneath the retina and in the vitreous he succeeded in obtaining lasting visual results.

HOLT (206) reports six cases in all of extraction of foreign bodies from the vitreous. In four the extraction was made by means of a Bradford magnet. In all except one the vision was restored to its normal condition. In one case the removal was affected by means of a Mathieu's forceps which was introduced into the vitreous, and search made for the body until it was found. In this instance the vision was not good, but the form of the eyeball was preserved. BURNETT.

In the case reported by SCHWARTZSCHILD (207) a man of twenty-eight received a violent injury to the head when he was a boy of eight. When seen by Schwartzschild there was a sinking of the eye deep in the orbit, which was changed into an exophthalmos when the patient leaned the head forward and downward. Vision was good and fundus normal. No pulsation or bruit. The writer does not think that the proptosis is due to vascular disturbance, owing to

the entire absence of the symptoms of such condition ; but holds that it is most likely due to air which enters the orbit from a communication with the frontal sinus caused by the injury.

BURNETT.

XXI.—OCULAR AFFECTIONS IN CONSTITUTIONAL DISEASE.

208. MAGNUS, H. Ophthalmological charts for academical teaching and private study. Breslau, 1892, I. The diagnosis of central disease of the optical apparatus.

209. KORTNEW. On cataract as a result of ergotism. *Wjestn. Ophthalm.*, 1892, No. 2.

210. HANSEN. Ocular diseases following influenza. *Hosp. Tidende*, p. 1229, Kjöbenhavn, 1891.

211. ANTONELLI. Neuritis optica retrobulbaris and papillitis from influenza. *Ann. di Ottalm.*, vol. xxi., 2–3, p. 119.

212. DEUTSCHMANN, R. Morbid changes in the eye in leukæmia. *Beiträge zur Augenheilk.*, 1892, part 4.

213. VOSSIUS, A. A case of ocular disease from lightning. *Beiträge zur Augenh.*, 1892, part 4.

214. v. HIPPEL, E. On debated questions connected with tumors of the pituitary body. *Virch. Archiv*, vol. cxxvi.

215. BOLTZ, R. A case of acromegaly with bitemporal hemianopsia. *Deutsch. med. Wochenschrift*, 1892, p. 634.

216. BULL, C. S. Report of a case of brain tumor, with autopsy. *Trans. Am. Oph. Soc.*, 1891.

217. MADER. Discussions of debated questions. *Wiener med. Blätter*, 1892, Nos. 4 and 6. (1.) Right sided hemianopsia of both eyes, caused by white softening of the right occipital lobe. (2.) Sarcoma of the right frontal lobe; visual disturbance; choked disc.

218. ZIEM. Exophthalmus and thrombosis of the cerebral sinus. *Wien. klin. Wochenschrift*, 1892, Nos. 26 and 27.

219. OLIVER, C. A. A clinical study of the ocular symptoms found in the so-called Mongolian type of idiocy. *Trans. Am. Oph. Soc.*, 1891.

220. SÄNGER. A study of nervous affections in the early stage of syphilis. *Jahrb. der Hamburger Staats-Krankenanstalten* Jahrg. 1890, part 1.

221. CHRIST, A. On debated questions in nuclear ophthalmoplegia. *Deutsches Arch. klin. Medicin*, vol. xlvi., parts 5 and 6.

222. BORTHEN, L. The local diagnostic conditions in unilateral, isolated, reflex iridoplegia. *Zehender's klin. Monatsbl. für Augenheilk.*, vol. xxx., p. 121. [Probably disease of the sphincter nucleus.]

223. REDLICH. On the peculiarities of reflex iridoplegia in progressive paralysis. *Neurol. Centralbl.*, 1892, p. 307.

224. GRADDY, L. B. Report of a case of blindness concomitant with la grippe — permanent (?) impairment of visual fields. *Oph. Rec.*, June, 1892.

225. DE SCHWEINITZ, G. E. A note on asthenopia and intranasal disease. *Med. News*, April 2, 1892.

226. RANNEY, A. L. Some prevalent errors relating to "eye-strain" as a cause of nervous derangement. *N. Y. Med. Jour.*, June 11–28, 1892.

227. BATTEN, R. D. Myopia the result of constitutional disease. *Ophthalmic Review*, vol. xi., 1.

These plates from the hand of MAGNUS (208) constitute, in form and contents, a splendid gift to both physicians and students. They cannot be too strongly recommended.

During a widespread epidemic of ergotism in the Government of Wjatka, KORTNEW (209) observed patients with two different kinds of visual disorders. One class complained of a transitory cloudiness of vision, when V fell to $\frac{5.0}{100}$ and less. This symptom lasted several minutes, but its occurrence varied greatly from several attacks daily to one in a month. It was present most commonly in connection with the more pronounced convulsive attacks. In patients of the second class, the visual failure was gradual and permanent. In both of them, lenticular opacities appeared and spread from the centre to the periphery. 37 such patients, from six to fifty-four years of age, were discovered in 500 cases of poisoning. In the younger patients the cataract matured in from 2 to 3 months, but in the adults it occupied from 8 to 12 months. Its extraction and the subsequent healing of the wound were not marked by any unusual incident, except a decided tendency to loss of vitreous. During the discission of semifluid cataracts Kortnew observed that absorption was very tedious, and in two cases, after each discission, severe attacks of convulsions, due to the ergot poisoning, set in. HIRSCHMANN.

ANTONELLI (211) reports two cases of optic-nerve disease following influenza. In the first instance, there was a gray-green discoloration of the papilla in both eyes. V = $\frac{1}{10}$ to $\frac{1}{15}$; visual field showed a slight limitation outwards, and was defective for red and green. Diagnosis: old retrobulbar optic neuritis. The second case was a marked example of bilateral neuro-retinitis. Both eyes were very myopic and vision was fingers counted at 1 *m*, while color-sense was entirely lost. Field of vision normal. In both cases the ocular symptoms showed themselves as the influenza disappeared and convalescence set in. DANTONE.

HIPPEL'S case (214) of sarcoma affecting the pituitary body in a woman presented the following symptoms: headache, amblyopia, transitory paralyses of ocular muscles, choked discs, and, later, optic atrophy.

BOLTZ (215), in view of the fact that the post-mortem examination of four cases of acromegaly demonstrates an enlargement of the pituitary body, conjectures that in his case there was also a tumor of the latter organ.

The peculiarity about BULL'S (216) case is that the patient a man of forty-five, had had no pain in the head at all except as he died. As regards the eyes, there was for a year or more before death an inequality in the size of the pupils. Nine months before death, there was a marked ptosis of the right upper lid and paresis of both internal recti. In the left eye the pupil was moderately dilated and immobile. There was crossed diplopia and the right image was lower. Moderate neuro-retinitis in the right eye with some small hemorrhages. Under treatment, all these symptoms disappeared. Later there was some vertigo. He died in a sudden attack and in delirium. The autopsy showed a tumor —glio-sarcoma—involving the corpus callosum and protruding downward from the roof of the lateral ventricle. There was no sclerosis of the nerve tissue in any part of the brain.

BURNETT.

OLIVER (219) has made extensive studies of the subjects in the Pennsylvania Institute for Feeble-Minded Children, and reports in this paper on the changes found in the Mongolian form of idiocy. He attributes the peculiar shape of the palpebral aperture to the form of the orbits and their relation to each other, and to the peculiar attachments of the ligaments inherent in the form of the skull. There are nearly always appearances of low forms of

neuro-retinitis and choroiditis. Other changes, both intra-ocular and extra-ocular, are due evidently to imperfect development.

BURNETT.

SÄNGER (220) reports six cases of syphilis in its early stages where marked anatomical changes had taken place in the nervous system, and cautions us not to imagine that the first manifestations of the disease are confined to the skin and mucous membranes alone. In the patients referred to he found retrobulbar neuritis, bilateral atrophy, and muscular pareses, which were first indicated by a decided concentric limitation of the field.

In CHRIST'S (221) case a gliosarcoma of the pineal gland had given rise to a trochlearis paresis.

BORTHEN'S (222) case is as follows: a sailor, æt. thirty-seven, was infected nineteen years before and suffered from pains in the head and vertigo associated with a unilateral, isolated reflex iridoplegia. Of the three possible explanations of these symptoms, viz., nuclear disease, affection of Meynert's fibres (as well as of those joining the two oculo-motor centres), and spinal irritation, the writer prefers the first.

REDLICH (223) has discovered that, in the case of those paralytics whose one eye shows a reflex iridoplegia while the pupil of the other still reacts to light, the former produces a consensual reaction upon the latter. The converse of this is, however, not true. This is explained by postulating a partial crossing of the pupillary fibres of the opticus outside the chiasma or the posterior commissure.

In GRADDY'S (224) case a woman in the third week of an attack of the grippe developed a sudden blindness, which after many fluctuations from better to worse finally settled down to a left hemianopsia. The optic disc had not changed its appearance in any important particular. The blindness is evidently due to an intracranial affection. BURNETT.

DE SCHWEINITZ (225) reports three cases of asthenopia with ametropia and heterophoria, in whom the correction of these anomalies failed to relieve the patient, and where the treatment of the rhinitis was followed by great benefit. BURNETT.

RANNEY (226) is a neurologist who has given much attention to "eye-strain" as a cause of nervous derangements. He here brings forward the question again and gives, in support of his opinion, the histories of eighteen cases in full. Among the con-

clusions at which he arrives are the following, which may be of interest.

Atropine mydriasis is the only condition for a scientific determination of refraction. Correction of muscular anomalies is of little or no value until errors of refraction have been carefully corrected. Eye-strain is a frequent cause, and perhaps the most important of all factors that tend to produce functional nervous disease.

He has never seen a case of typical sick-headache which was not associated with eye-strain. The commonest causes of nervous derangements are esophoria, hyperphoria, and hypermetropia. Exophoria and myopia are less frequently causes.

BURNETT.

BATTEN (227) imagines that high pulse tension and other vascular changes are a common cause of myopia; eighteen illustrative cases are described, the last being headed, "Myopia and chronic bronchitis in a child"! What a wonderful coincidence!

WERNER.

BOOK REVIEWS.

1. The Relations of the Visual Organ and its Diseases to the other Diseases of the Body and its Organs. (Die Beziehungen des Sehorgans, etc.) By Dr. MAX KNIES, Prof. University of Freiburg; J. F. Bergmann, Wiesbaden, 1893; 484 large octavo pages, with many illustrations in the text, and a good index.

1. This book is one of the most important and interesting treatises that have appeared since the *Handbuch* of Græfe-Sämisch. It treats of the subject in the same way, only more extensively, than Förster did 16 years ago. The advances in this department during the last 16 years have been enormous, especially in neurology with which ophthalmology is so intimately connected. The author gives in detail all the anatomical and physiological facts that have any bearing on his subject, thus furnishing invaluable demonstrations and explanations of the numerous and intricate pathological phenomena which the eye exhibits in other diseases of the body. The occurrence of ocular symptoms, their detection and significance in diseases of the nervous system, occupies 255 pages a little more than half of the whole book. This most important department is analyzed and presented with such clearness and thoroughness that no neurologist, oculist, or general practitioner will resort to it without finding what is known on the subject. Even the surgeon will receive most valuable information as to the origin and significance of ocular symptoms produced by brain lesions for which the skull should, or should not, be opened. The connections of eye diseases with those of other organs of the body and general diseases are discussed with due thoroughness according to their importance. The style of the book is concise and eminently clear. The author calls the work under consideration the second part of his treatise of ophthalmology (J. B. Bergmann, 2d edition), and a supplementary volume to every text-

book of internal medicine or ophthalmology. We can heartily endorse this statement and would add that the book ought to be translated into English. H. K.

2. **Les Maladies des Yeux dans leurs Rapports avec la Pathologie Générale.** By Dr. EMILE BERGER; G. Masson, Paris, 1892; 457 pages in large octavo, with 43 drawings in the text, and an index. This book is very similar to the previous, and forms a course of lectures delivered by the author (a native of Austria, formerly lecturer at the University of Graz) at Paris, collected by Dr. R. DE SAINT-CYR DE MONTLAUR, revised by the lecturer. The first 70 pages are devoted to a general description of the anatomy, physiology and pathology of the ocular nervous apparatus so far as they give rise to symptoms not exclusively ocular: intra-cerebral and extra-cerebral paralyses of the motor nerves of the eye, centre of co-ordinate movements, conjugate deviation, paralysis of convergence, pupillary reaction, localization of the central lesion according to the functional disturbance of the eyes, and a variety of other conditions. The special part describes the diseases which produce ocular troubles in disease of the nervous system (80 pages), the skin, the ear, the nose and its accessory cavities (39 pages, very good), the mouth and the alimentary canal, the respiratory organs, the blood, the urinary and genital organs, germ diseases, poisoning with a multitude of substances mostly medicinal, trade diseases, hereditary influences, and senility.

We need not say that a book with so extended a scope will be most welcome both for information and reference. It has the advantage over Knies' book of being written in French, a more international language than German. H. K.

3. **Introduction into Ophthalmology** (Einführung in die Augenheilkunde). By Dr. J. HIRSCHBERG, Professor University Berlin, I. Half. Geo. Thieme, Leipzig, 1892; large octavo, 234 pages, very well printed, with 112 drawings. The well-known author describes in this first half of a compendium of ophthalmology, in his original and independent manner: (1) The remedies used in ophthalmology. (2) The operations on the eye (antisepsis (very detailed), anæsthesia, general technique). (3) The examination of the disturbances of vision (10 pages). (4) Refraction, dioptrics and designation of spectacles ("The refractive power of a lens of one meter focal distance has been chosen as a

unit and called dioptry. The idea originated with Nagel, the name with Monoyer; it was introduced by Donders, and objected to by Hasner, Mauthner, and myself"); E. M., and H. (5) Accommodation. The author believes that the latent H which young people show on functional examination, disappears when, in the dark room, they hold their eyes open unconcernedly, and an oculist, skilled in the determination of the refractive state, will detect the whole amount of H without the use of atropine (p. 159). The reviewer, who as long as he has practised ophthalmology has used the direct method as routine work in every case of refraction, is of opinion that in most cases the ophthalmoscope alone suffices, but in some the use of a mydriatic (a 3 or 4 per cent. solution of homatropine) brings out a higher degree of H. (6) Dioptrics. This chapter is excellent and complete, essentially according to the famous § 9 in Helmholtz's *Physiologische Optik.*

Hirschberg's treatise will surely be read with interest and benefit by every oculist. If the author treats the remainder of his subject with the same thoroughness as his chapter on dioptrics, his intelligent reader will be well prepared to understand all ophthalmological literature and to solve new problems which do not lie beyond the sphere of our present civilization. H. K.

4. **A Manual of Clinical Ophthalmology.** By H. F. HANSELL, M.D., and J. H. BELL, M.D. 231 pages, small octavo, well gotten up; 120 illustrations. P. Blakiston, Son, & Co., Philadelphia, 1892. This is a very well written, elementary treatise, adapted to the college student and physician who does not want to limit his practice to diseases of the eye. Price $1.75. H. K.

5. **Ophthalmic Diseases and Therapeutics.** By A. B. NORTON, M.D., Professor of Ophthalmology in the (homœopathic) College of the New York Ophthalmic Hospital, etc. Boericke & Tafel, Philadelphia, 1892; 555 large octavo pages, well printed on good paper, with 53 wood-cuts and 12 small colored ophthalmoscopic pictures. This volume has peculiar features. The organic diseases of the eye are described as in the ordinary text-books, and well described, but many subjects of importance—for instance, the whole of refraction and accommodation, both physiological and practical—are omitted. Functional examination (acuteness and field of vision, color-perception, light-sense, eyeball tension, etc., do not seem to have been mentioned)

and even the ophthalmoscope are not described. The two plates of ophthalmoscopic drawings with one page of explanation of the chromo-lithographs appended to the volume appear to have no connection with the text. The illustrations are all reproductions. In the chapter on glaucoma we find beside glaucoma absolutum, glaucoma acuta, gl. consecutiva, gl. chronica. The distinguishing feature of the book is the internal treatment of eye diseases. After each disease and its regular treatment, there is, in alphabetical order, a string of internal homœopathic remedies, which are collected and reproduced in Part II. of the work, the ophthalmic therapeutics, taking up one third of the whole space. H. K.

6. **Text-Book of Ophthalmology.** By Dr. E. FUCHS, Prof. University of Vienna. Authorized translation from the second (newest) German edition, by Dr. A. DUANE, of New York. 788 closely printed large octavo pages, with numerous illustrations, mostly original. D. Appleton & Co., New York, 1892. Price $4. This is the English translation of the German text-book, which we have reviewed in a former volume of these ARCHIVES. We can only repeat that it is, in our opinion, one of the very best text-books of ophthalmology, and we desire to add that Dr. Duane has done his work admirably well. He deserves credit and thanks for having made the celebrated Vienna professor's book accessible to English readers. H. K.

7. **Transactions of the Ophthalmological Section of the Am. Med. Assoc.**, at the 43d Meeting, held at Detroit, Mich., June, 1892. This is a neat volume of 367 small octavo pages, containing the numerous papers and discussions of the meeting, reprinted from the *Journal of the Am. Med. Assoc.* It is a great convenience for oculists to have these papers, which, almost without exception, are valuable contributions to ophthalmology, united in a handy volume.

8. **A Handbook of the Diseases of the Eye and their Treatment.** By H. R. SWANZY. Fourth edition, 1892. P. Blakiston, Son & Co., Philadelphia. 580 octavo pages. Price $3.

We welcome with pleasure this new edition of one of the standard treatises on the eye. It deservedly continues to be one of the most popular text-books for students and practitioners. The competent author, whose good judgment is apparent on every page, is most zealous in keeping up with the progress of science, of which this new, carefully revised edition bears testimony. H. K.

MISCELLANEOUS NOTES.

APPOINTMENTS.

Prof. HERMANN KUHNT, M.D., has been appointed to the chair of Ophthalmology at the University of Breslau, Prussia, and Dr. GRÖNOUW, lecturer (Privatdocent) at the same university.

Dr. R. O. BORN has been advanced from assistant surgeon to surgeon at the New York Ophthalmic and Aural Institute. Many advances from clinical assistant to assistant surgeon have been made at this and other ophthalmic hospitals of New York. The names of the gentlemen are found in the annual reports of these hospitals. All these institutions show a great deal of activity, not only in charitable but in didactic and scientific work.

The same can be said of the institutions of Philadelphia, where Dr. G. E. DE SCHWEINITZ, lecturer at the University of Pennsylvania, has been appointed Clinical Professor of Ophthalmology at Jefferson Medical College, with ample clinical and teaching facilities. Similar development exists all through the country. THE ARCHIVES would gladly record the names if the gentlemen would only favor them with the information.

SOCIETY MEETINGS.

The meeting of the AMERICAN MEDICAL ASSOCIATION will take place at Milwaukee, Wis., on June 6th, 7th, 8th, and 9th. President of the ophthalmological section, Dr. RISLEY, Phila.; secretary, Dr. GRADLE, Chicago.

The PANAMERICAN MEDICAL CONGRESS will be held at Washington, D.C., September 5th–8th. Dr. J. J. CHISOLM, of Baltimore, the president of the ophthalmological section, cordially invites all physicians that take an interest in ophthalmology, to attend.

Fig. 1.

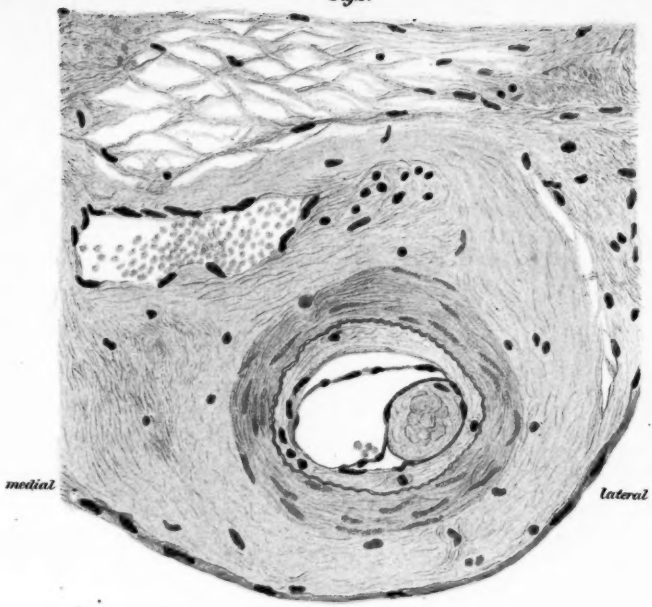


Fig. 2.

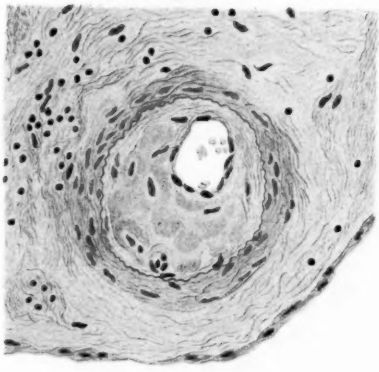
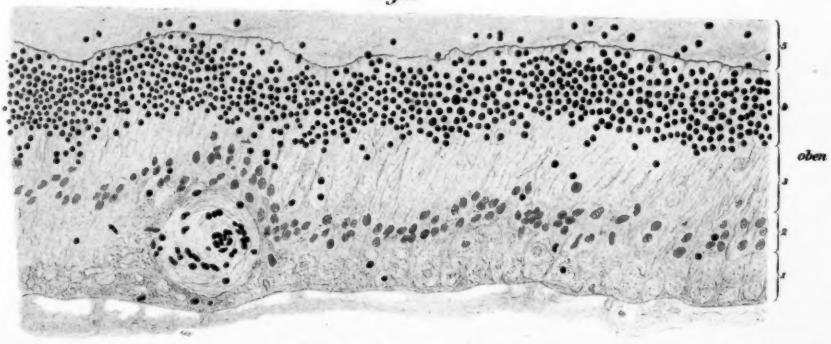


Fig. 3.



Verlag v. J.F. Bergmann, Wiesbaden. Dr. Elschnig ad nat. del. Lith. Anst. v. C. Hirst, Leipzig.

Fig. 4.

Fig. 5.

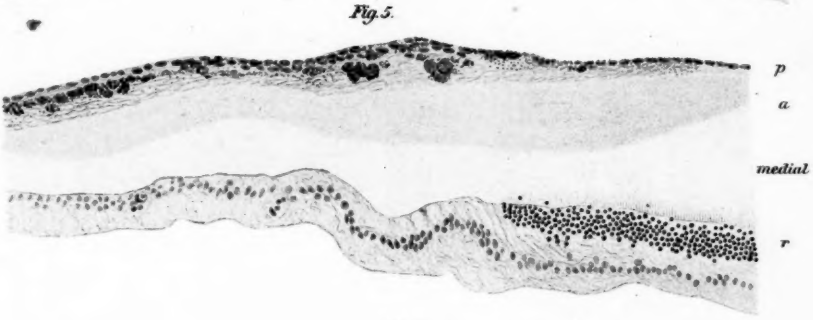


Fig. 6.

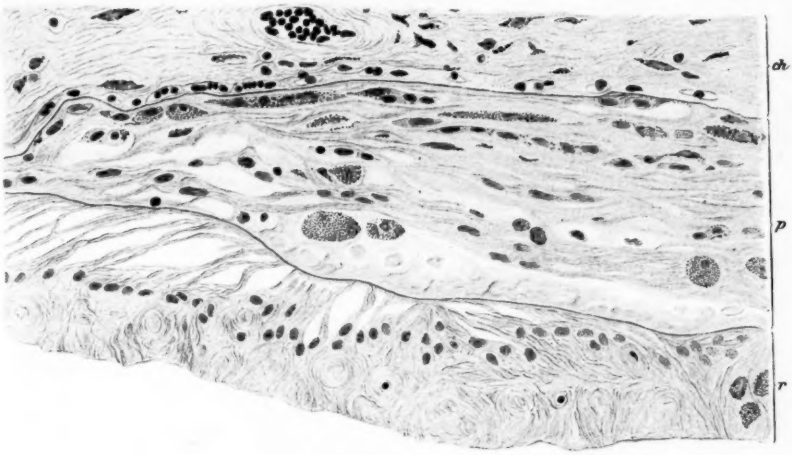


Fig. 7.

Verlag v. J. F. Bergmann, Wiesbaden. Dr. Elschnig ad. nat. del. Lith. Anst. v. C. Först, Leipzig.